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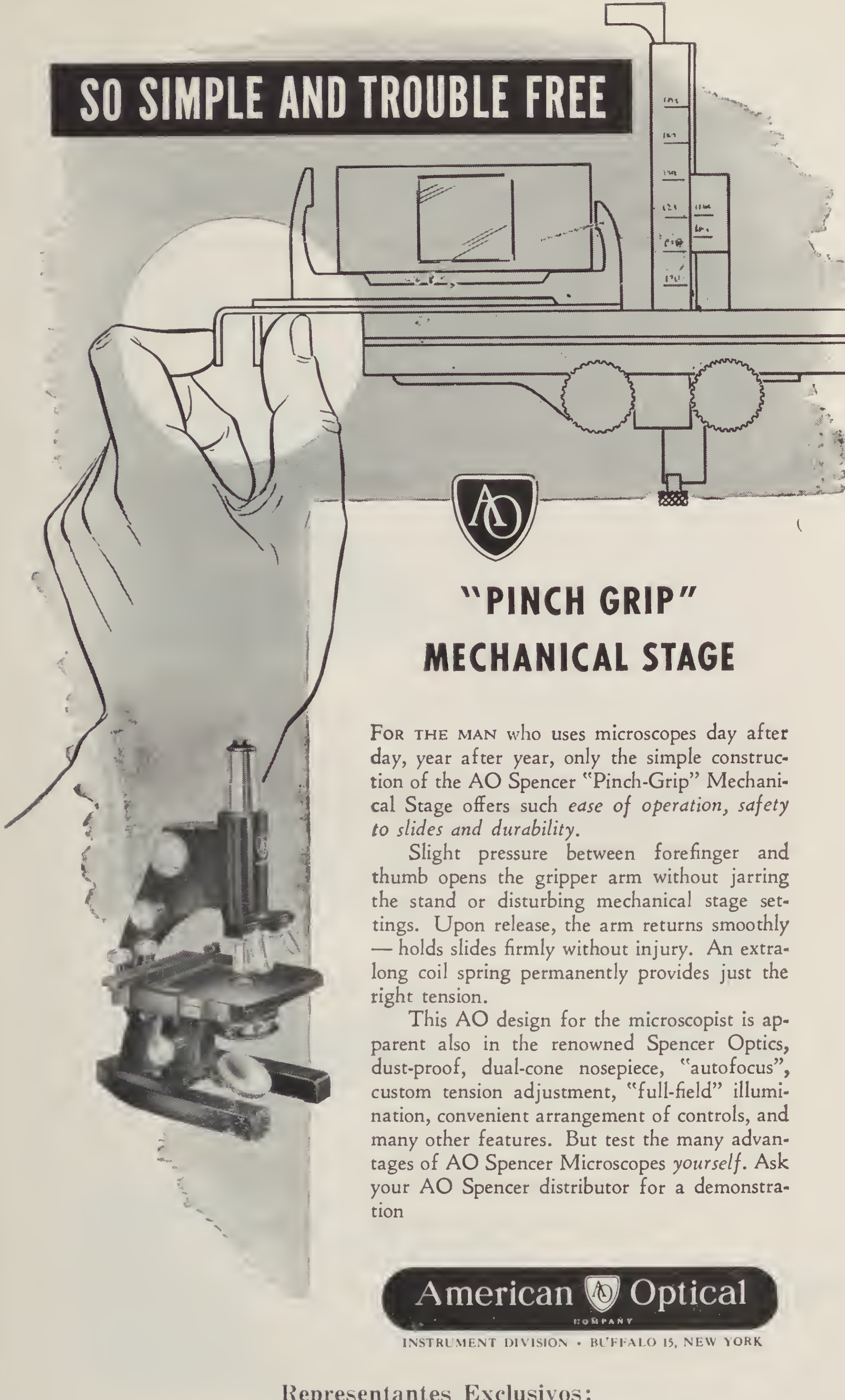
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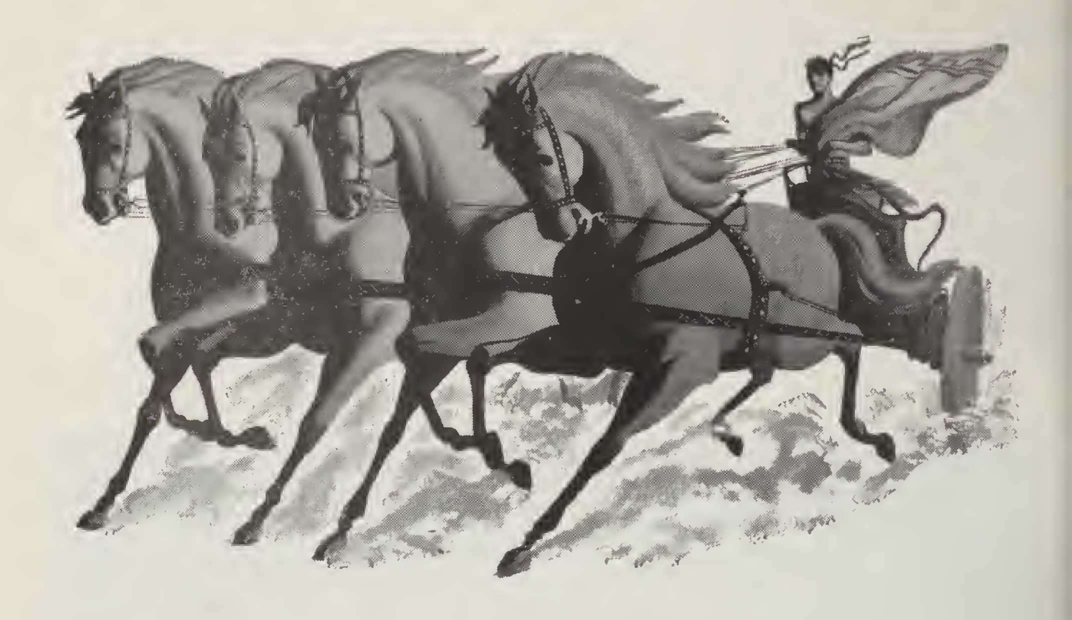
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ISONIAZID BY THE INTRATHECAL ROUTE IN THE THERAPY OF TUBERCULOUS MENINGITIS

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This study was undertaken in order to determine the usefulness of isoniazid by the intrathecal route in patients with tuberculous meningitis. Our aims were to determine (1) the dosage and manner of using it: (2) toxic reactions; (3) effectiveness. We believe that we have learned, roughly, how to use the drug by the intrathecal route and that toxic reactions, if any, are minimal. We still have to learn how effective is this form of therapy. Many authorities[1] believe intrathecal therapy is no longer necessary in tuberculous meningitis. We believe this is true in most cases, but we are not certain that therapy by the intrathecal route should be completely discarded.

MATERIAL AND METHODS

Preparation of Isoniazid:

Lyophilized isoniazid was supplied by courtesy of Panray Corporation. It was diluted to make a 2% solution by adding 50 ml. of sterile physiological saline solution to a 1 gram vial of lyophilized isoniazid.

Storage:

The solution of isoniazid may be stored at room temperature where it remains stable for months.

From the Department of Pediatrics of the Sanatorio Alejandro Ruiz Soler✧, Bayamón District Hospital,❖ San Juan City Hospital* and the School of Medicine of the University of Puerto Rico. This study was aided by grants from the Committee on Medical Research of the American Trudeau Society, Medical Section of the National Tuberculosis Association.

Dosage and mode of administration:

A dosage of 10 mg. was used at first; later it was increased to 20 mg. injected by the intrathecal route, once daily in the lumbar region. Doses of 10 mg. have been injected in the cistern without ill effects. The doses were not calculated on a body weight or age basis; instead, an attempt was made to administer the highest dose compatible with safety. Italian authors have administered isoniazid intrathecally in doses of 1.5 mg/kg for infants and 1 mg/kg for children.[2]

Our first group of patients received the 2% isoniazid solution, 0.5 to 1ml., diluted in 5 ml. of N saline or cerebrospinal fluid; however as we became more familiar with the drug we discovered that it could be administered as the 2% solution without further dilution. In this way it has been invariably well tolerated.

Selection of Patients:

The patients were chosen from a group of children with tuberculous meningitis admitted to the Bayamón District Hospital, the San Juan City Hospital and the Sanatorio Alejandro Ruiz Soler. At first it was administered only to patients who were running a downhill course despite our usual forms of therapy, including isoniazid orally 7-10 mg/kg, streptomycin intramuscularly 1 gm. daily and often streptomycin by the intrathecal route 50 to 100 mg. daily. Our routine has been reported previously.[3] When we became convinced that isoniazid by the intrathecal route was harmless we began to administer it to less seriously ill patients and later, instead of streptomycin, by the intrathecal route. Now we have abandoned streptomycin by this route because we have observed that 50% of the patients, who receive streptomycin into subarachnoid space, become deaf.

The diagnosis of tuberculous meningitis in these patients was based on the following criteria: (1) physical findings typical of tuberculous meningitis; (2) positive tuberculin reaction; (3) abnormal spinal fluid consisting of low glucose, high proteins and increased cells, mainly lymphocytes; (4) evidence of tuberculosis in the chest x-ray. The presence of acid fast bacilli in the cerebrospinal fluid was not considered essential to make a diagnosis of tuberculous meningitis and therapy was administered regardless of negative bacteriological reports. The severity of the tuberculous meningitis has been graded as first stage (early); second stage (moderately advanced), and third stage (late).[4] All the surviving patients have been followed until October, 1954, and all had normal cerebrospinal fluid before or at the completion of 1 year of

chemotherapy. All graphs do not show this return of the cerebrospinal fluid to normal because of lack of space.

CASE REPORTS

The following case reports illustrate how the drug is tolerated, its effects upon the cerebrospinal fluid chemistry and cytology and the clinical course of the patient.

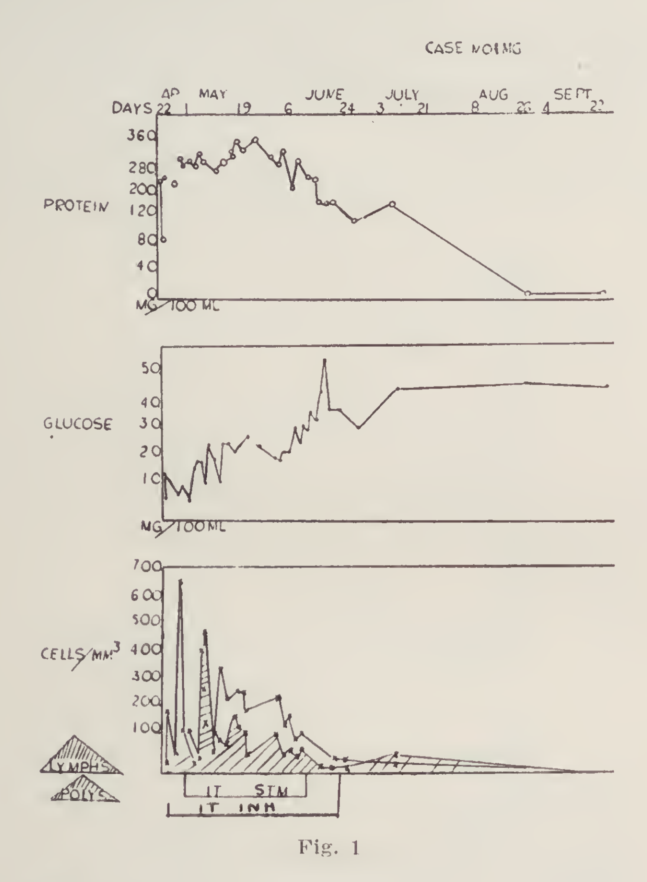


Fig. 1

Case #1, (M. G.), Fig. 1

This 11 month-old male infant was admitted to the Sanatorio Alejandro Ruiz Soler on February 13, 1953, with primary pulmonary tuberculosis. On April 22, 1953, he developed early tuberculous meningitis without neurological signs, but with acid fast bacilli in the cerebrospinal fluid. On April 23, 1953, therapy for tuberculous meningitis was started. Daily intrathecal isoniazid 10 mg, intramuscular streptomycin, oral PAS, and isoniazid were started immediately. On the second week of treatment, intra-

thecal streptomycin was added in doses of 50 mg. daily. During this therapy the patient lapsed into second stage meningitis and became deaf and blind. Intrathecal streptomycin was discontinued on June 11, 1953; at the same time, intrathecal isoniazid was increased to 20 mg. daily and continued until June 23, 1953, when the return of cerebrospinal fluid glucose to normal permitted discontinuing intrathecal therapy. Gradual regression of cerebrospinal fluid abnormal findings has been observed. The electroencephalogram which was very abnormal during the acute stage is returning to a more normal pattern. The patient has recovered from deafness and blindness, and appears cheerful.

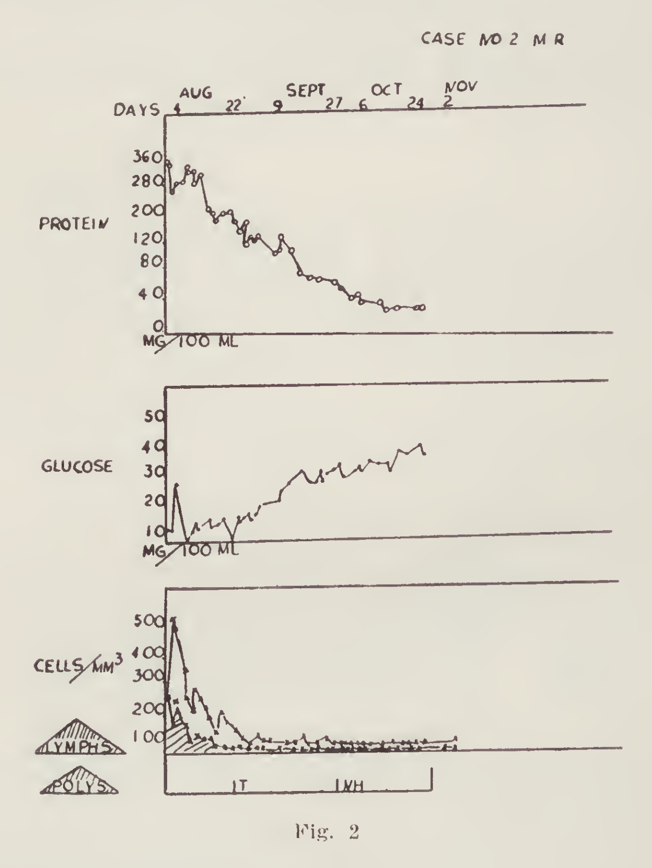


Fig. 2

Case #2, (M. R.), Fig. 2

This 6 year-old girl was admitted to Fajardo District Hospital, Fajardo, Puerto Rico on September 23, 1952, with moderately advanced tuberculous meningitis. She was treated with streptomycin intramuscularly and intrathecally and PAS orally; improved and was discharged on January 19, 1953.

On February 25, 1953, she was readmitted with a relapse of the tuberculous meningitis. This time she was treated with streptomycin intramuscularly and intrathecally and PAS and isoniazid orally. She improved slowly and intrathecal therapy was discontinued on May 4, 1953. She was transferred to our Hospital on June 24, 1953. At this time she was deaf, but had no other neurological signs except ataxia. Therapy with streptomycin intramuscularly, isoniazid orally and PAS was continued. In spite of this she began to lose weight and her cerebrospinal fluid became very abnormal with very low glucose and high protein. On August 4, 1953, daily therapy with isoniazid by the intrathecal route, 20 mg. daily, was started. No toxic reactions were observed. There was improvement in the general condition of the patient. On October 28, 1953, intrathecal therapy was discontinued. The patient is well except for deafness.

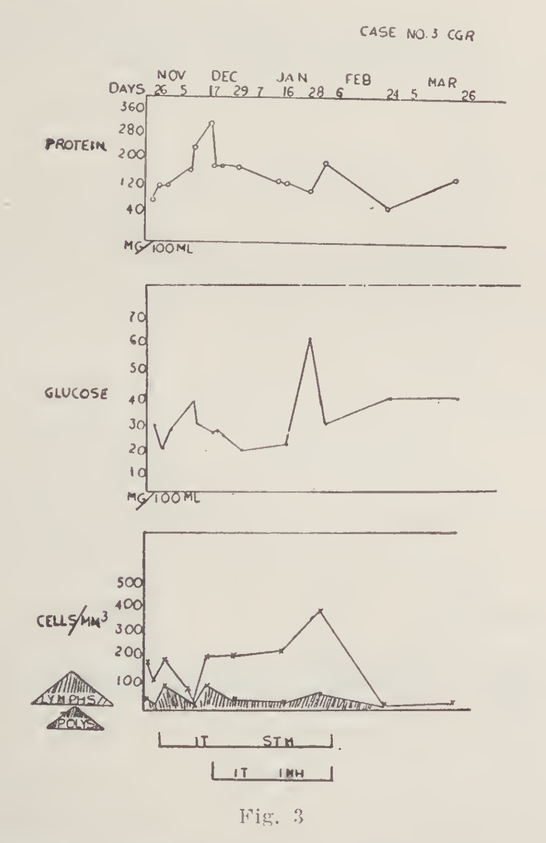


Fig. 3

Case #3, (C. G. R.), Fig. 3

A 2 year-old male was admitted on November 26, 1952, to Bayamón District Hospital, Bayamón, Puerto Rico with second

stage tuberculous meningitis. Routine therapy of tuberculous meningitis was started on November 28, 1952. During therapy he developed gross tremors, became more spastic, irritable, deaf and blind. Cerebrospinal fluid glucose values were persistently low. On December 19, 1952, while the patient was still receiving intrathecal streptomycin, therapy with isoniazid by the intrathecal route was added in doses of 10 mg. daily. No toxic effects were observed. The intrathecal isoniazid and intrathecal streptomycin were discontinued on February 2, 1953, when a slight improvement began to occur and the cerebrospinal fluid glucose values began to rise.

In May, 1953, striking changes in the general condition of the patient were observed. The patient could see, hear, was not irritable, and began to talk. This improvement together with weight gain and normal temperature has persisted.

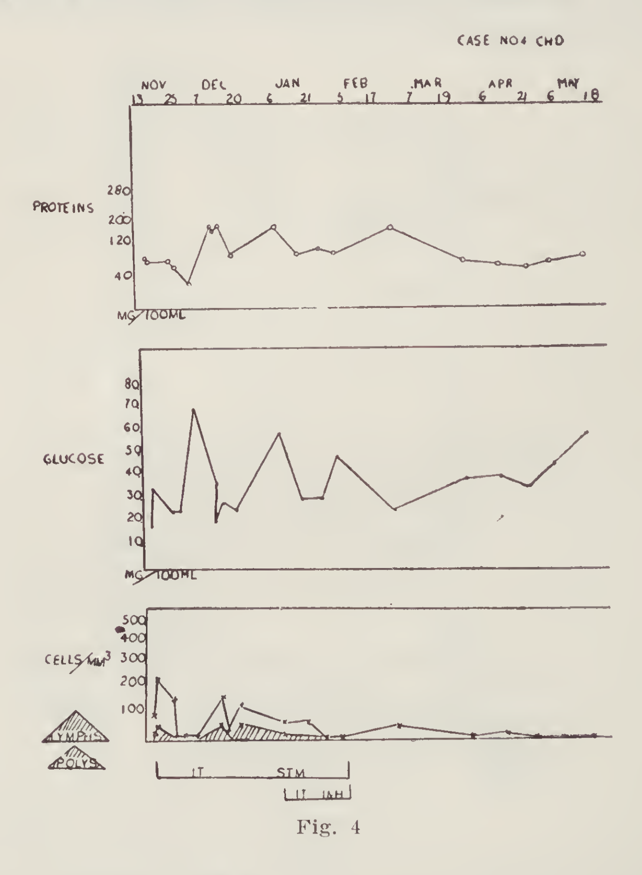


Fig. 4

Case #4, (C. H. D.) Fig 4

A 9 year-old male was admitted on November 13, 1952, to Bayamón District Hospital, Bayamón, Puerto Rico with third stage tuberculous meningitis. Routine therapy of tuberculous meningitis

was started the following day. The patient continued to be in a semicomatous stage with spasticity of the extremities gradually increasing. The cerebrospinal fluid glucose was persistently low. On January 5, 1953, while still receiving intrathecal streptomycin, therapy with isoniazid 10 mg. daily by the intrathecal route was started. No toxic effects were noted. On February 2, 1953, because the cerebrospinal fluid glucose was increasing the intrathecal therapy with streptomycin and isoniazid was discontinued.

In March, 1953, a marked improvement in the general condition of the patient was noted; he could sit up, could hear loud voice (he had a mastoidectomy before present illness) and talked. Vision was normal. He still had a flexion contracture of the right knee which disappeared almost completely four months later. At present he is doing well except for partial deafness.

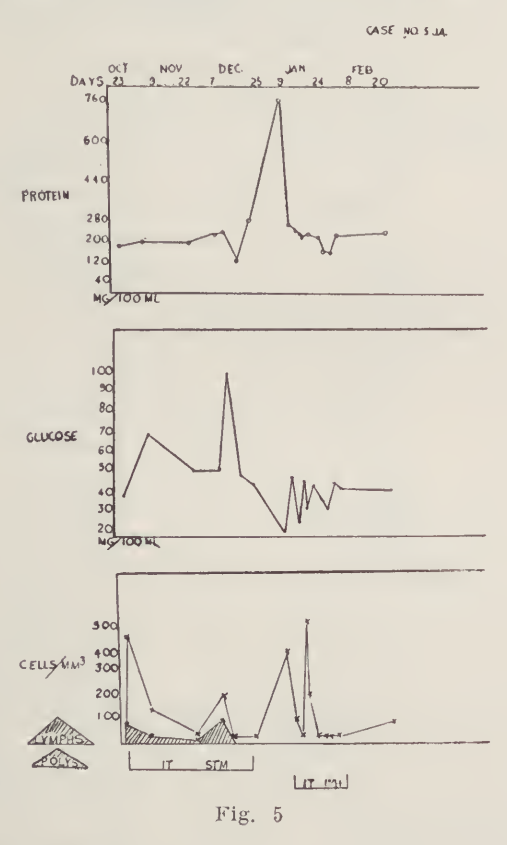


Fig. 5

Case #5, (J.A.P.), Fig 5

A 2 year and 5 month-old male was admitted to Bayamón District Hospital, Bayamón, P. R. on November 23, 1952, with second stage tuberculous meningitis. Routine therapy of tuberculous meningitis was started on the day of admission. No improvement

was noted; the patient was stuporous, had spasticity of the extremities, and convergent strabismus. On December 19, 1952, intrathecal streptomycin was discontinued because of increasing cerebrospinal fluid glucose values. On January 7, 1953, he had a recrudescence of the disease, manifested by increasing meningeal signs and more abnormal cerebrospinal fluid. On January 10, 1953, therapy with intrathecal isoniazid 10 mg. daily was begun. No toxic effects were noted. After January 29, 1953, definite improvement was noted. Nuchal rigidity had disappeared and the cerebrospinal fluid was less abnormal. The intrathecal isoniazid was discontinued on February 2, 1953. He continued to improve and at present is doing well except for moderate weakness of the lower extremities.

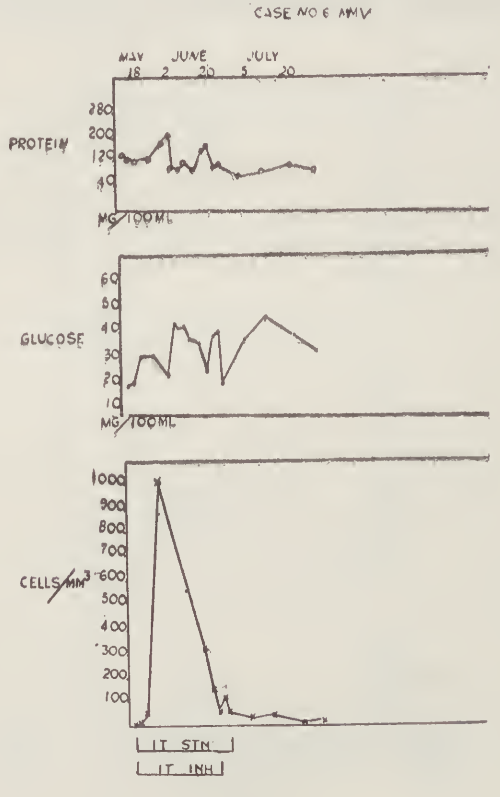


Fig. 6

Case #6, (M. M. V.), Fig 6

An 18 month-old female was admitted to Bayamón District Hospital, Bayamón, Puerto Rico on May 18, 1953, with second stage tuberculous meningitis. Routine therapy for tuberculous meningitis was started on the day of admission. Next day therapy with intrathecal isoniazid was started together with the intra-

thecal streptomycin. The first dose of intrathecal isoniazid was 100 mg. of the 2% solution by a mistake; but thereafter only 10 mg. was administered daily. Neither dose produced any toxic reaction. The patient showed a moderate initial improvement, became more alert and the papilledema noted on admission disappeared. However, the patient was deaf, mute, blind, irritable. The intrathecal isoniazid was discontinued on June 21, 1953, and the intrathecal streptomycin on June 30, 1953. The general condition remained stationary until August, 1953, when the patient was no longer irritable and began to hear and talk. In September, 1953, the improvement continued and the patient began to see again. At this time she was discharged to complete treatment at home. At present she is doing well except for recurrence of deafness on the seventh month of therapy, one month after streptomycin by the intramuscular route was discontinued.

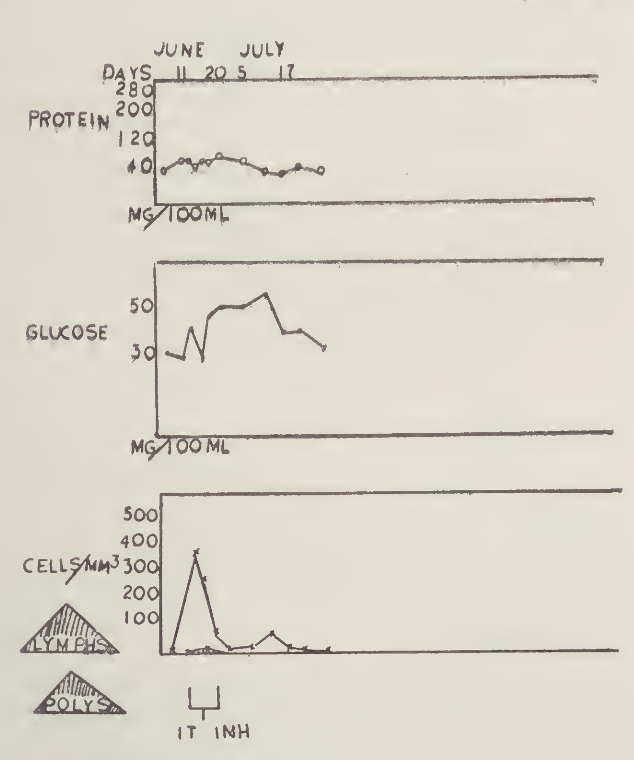


Fig. 7

Case #7, (R. C. R.), Fig. 7

A 24 month-old male was admitted to Bayamón District Hospital, Bayamón, Puerto Rico on May 16, 1953, with a gastroenteritis. On June 5, 1953, a miliary tuberculosis was discovered.

Therapy with streptomycin by the intramuscular route accompanied by oral isoniazid 10 mg/kg of body weight was immediately started. Cerebrospinal fluid chemistry suggested a tuberculous meningitis; however, a diagnosis of first stage tuberculous meningitis was not made until June 13, 1953, when accurate cell counts were obtained. At this time therapy with intrathecal isoniazid 20 mg. daily was started. No toxic reactions were observed. On June 21, 1953, intrathecal isoniazid was discontinued. The patient has continued to improve and except for slight drowsiness there have been no abnormal neurological signs.

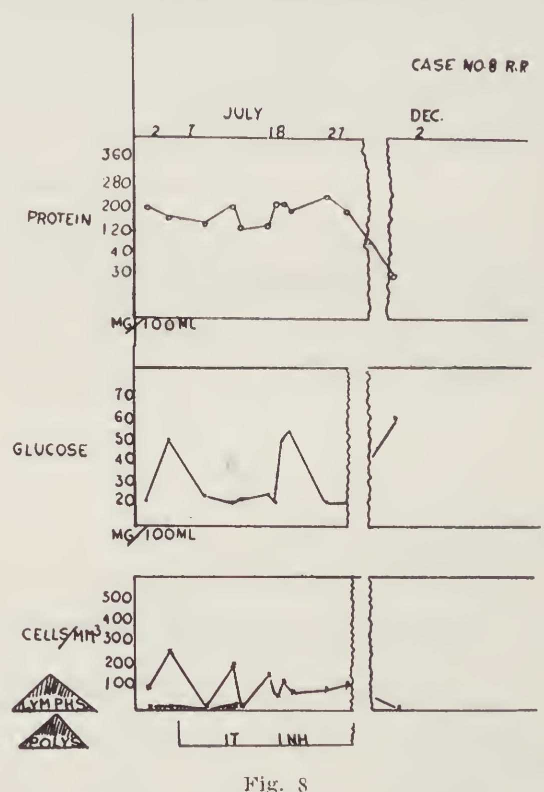


Fig. 8

Case #8, (R. P.) Fig 8

An 8 year-old female was admitted to Bayamón District Hospital, Bayamón, Puerto Rico on July 2, 1953, with second stage tuberculous meningitis. The following day therapy with isoniazid orally and streptomycin by the intramuscular route was started. She became stuporous, had spiking fever, and developed increasing neurological signs (positive Brudzinski and Kerning which had not been noted previously). On July 7, 1953, therapy with intrathecal isoniazid, 20 mg. daily was started. No intrathecal streptomycin was administered. A remarkable improvement took place;

within a 13 day period, she began to eat spontaneously and neurological signs disappeared except for slight nuchal rigidity. Intrathecal isoniazid was discontinued after 40 days of treatment. The patient has no sequelae and is well.



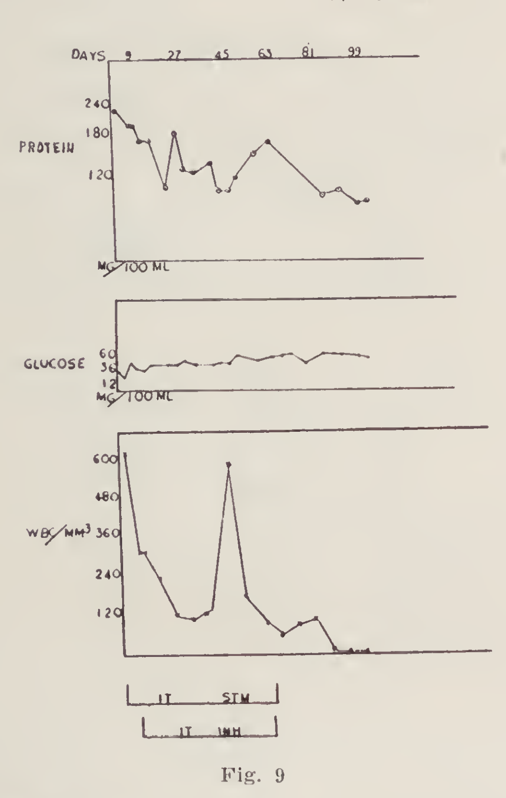


Fig. 9

Case #9, (R. M.), Fig 9

A 3 year-old boy was admitted to San Juan City Hospital, San Juan, Puerto Rico on April 23, 1953, with second stage tuberculous meningitis. Therapy for tuberculous meningitis was started on the day of admission, including streptomycin by the intrathecal route 100 mg. daily. At this time acid fast bacilli were cultured from the cerebrospinal fluid. On the 3rd. day of therapy the patient's condition was worse: he lapsed into third stage of tuberculous meningitis and the cerebrospinal fluid was more abnormal. On April 27, 1953, the dose of streptomycin was decreased to 50 mg. daily and intrathecal isoniazid 10 mg. daily was added. Two days later he was more alert and responsive. No toxic effects due to the intrathecal isoniazid were observed. By May 3, 1953, the spasticity was not present and steady improvement

was noted. On June 23, 1953 intrathecal therapy with isoniazid and streptomycin was discontinued in spite of which improvement has been steady. The patient has no sequelae and is doing well.

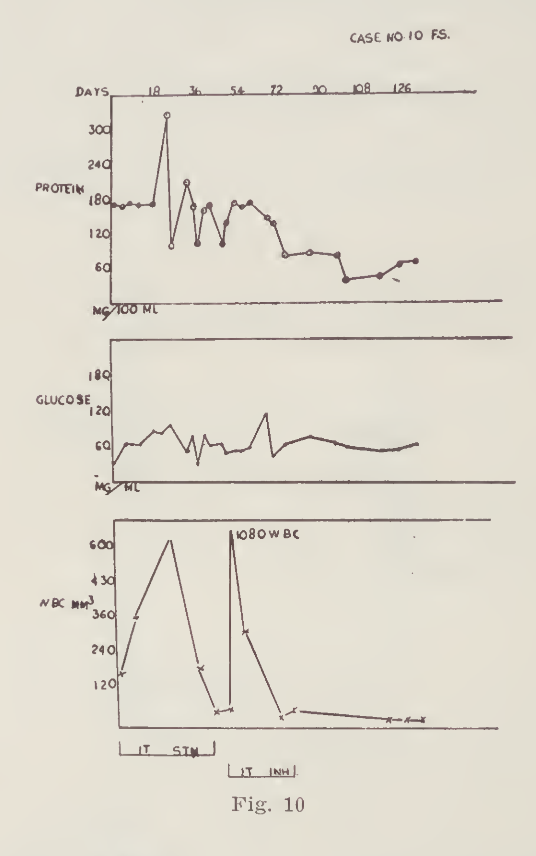


Fig. 10

Case #10, (F. S.), Fig. 10

A 4 year-old boy was admitted to San Juan City Hospital, San Juan, Puerto Rico on October 27, 1952, with late stage tuberculous meningitis. Therapy with streptomycin, isoniazid and PAS orally and intrathecal streptomycin was started on October 27, 1952. After 40 days of intrathecal streptomycin no improvement was noted. No signs of spinal block were present. The patient was spastic, unconscious and deaf. Streptomycin by the intrathecal route was discontinued and therapy with intrathecal isoniazid was started on December 16, 1952, 10 mg. daily. No toxic effects were observed. Six days later the patient became alert and conscious. Spasticity began to decrease and improvement was steady. Isoniazid by the intrathecal route was discontinued on January 13, 1953. At present the patient is doing well except for deafness and mental retardation.

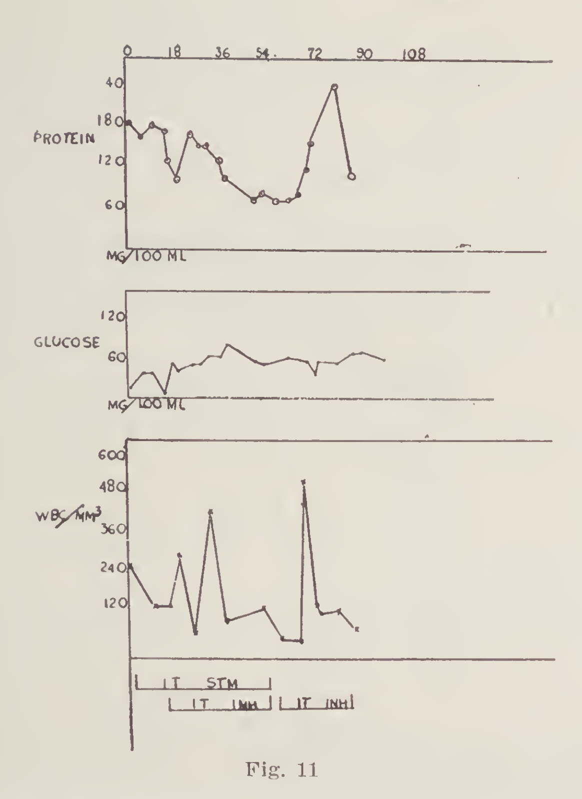


Fig. 11

Case #11, (R. A.), Fig. 11

A 1 year-old boy was admitted to San Juan City Hospital, San Juan, Puerto Rico on March 30, 1953, with late tuberculous meningitis. Therapy for tuberculous meningitis with intramuscular and intrathecal streptomycin, oral PAS, and isoniazid was started on March 31, 1953. No improvement was noted. On April 21, 1953, therapy with intrathecal isoniazid, 10 mg. daily, was started. Six days later a moderate improvement was noted. On May 21, 1953, the intrathecal therapy was discontinued. At this time the general condition had not improved although the cerebrospinal fluid findings were stabilized. Because of stationary conditions, intrathecal isoniazid was started on May 27, 1953, and intrathecal streptomycin was added on June 13, 1953. There was no further change and all intrathecal medication was discontinued on June 23, 1953. On June 29, 1953 the patient became worse and developed signs of bacteremia and urinary retention. He did not improve despite therapy with all available antibiotics including penicillin, sulfadiazine, oxytetracycline, and chloramphenicol. On July 25, 1953, the patient died. Autopsy was not performed.

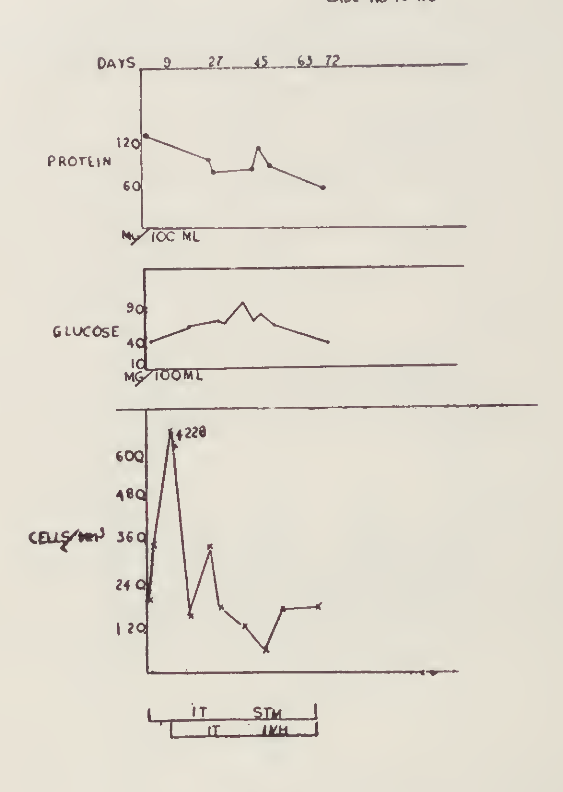


Fig. 12

Case #12, (N. S.), Fig. 12

A 2 4/12 year-old girl was admitted to San Juan City Hospital, San Juan, Puerto Rico on April 23, 1953, with moderately advanced tuberculous meningitis. Routine therapy for tuberculous meningitis was started on the day of admission including 50 mg. of streptomycin daily by the intrathecal route. The patient did not improve.

On April 29, 1953, therapy by the intrathecal route with isoniazid 10 mg. daily was added. No toxic effects related to intrathecal isoniazid were noted. On June 20, 1953, streptomycin intrathecally was increased to 100 mg. daily. On June 23, 1953, all intrathecal therapy was discontinued because the cerebrospinal fluid was returning to normal. However, the patient became worse, had frequent convulsions and died on June 30, 1953. Autopsy was not performed.

RESULTS

From the effects observed in these patients the following general statements can be made:

Effect upon the cell count of the cerebrospinal fluid

Although in some instances an increase in polymorphonuclear cells in the cerebrospinal fluid has been observed, the rule is that no remarkable change occurs unless streptomycin is administered simultaneously by the intrathecal route.

Effect upon the cerebrospinal fluid proteins

Usually a moderate transient increase in the protein is observed.

Effect upon the cerebrospinal fluid glucose

No rapid change in the cerebrospinal fluid glucose occurs coinciding with the improvement in the general condition of the patient.

Effect upon the tuberculous meningitis

Most of the patients improve slowly without showing the toxic reactions seen with intrathecal streptomycin such as loss of deep tendons reflexes, fever, irregularity of respiration and convulsions.

No immediate dramatic results have been observed in the clinical condition of the patients: however, in some patients whose clinical course was stationary or downhill a gradual but remarkable improvement occurred. We do not know if this improvement might have occurred if isoniazid had not been administered by the intrathecal route. In order to evaluate this form of therapy more accurately we have begun a controlled study of three groups using: (1) No intrathecal therapy; (2) isoniazid by the intrathecal route; (3) isoniazid and streptomycin combined by the intrathecal route.

Our observations may be summarized as follows:

Twelve children suffering from tuberculous meningitis were treated with isoniazid by the intrathecal route. Seven (cases 1, 3, 4, 6, 9, 11, 12) received streptomycin and isoniazid simultaneously by the intrathecal route. Five (cases 2, 5, 7, 8, 10) received the isoniazid alone or following a course of streptomycin.

The ages of the patients ranged between 11 months and 9

years. Two had early tuberculous meningitis, seven were moderately advanced and three had late meningitis.

Ten patients have survived for more than 14 months and are doing well, including two who recovered completely from deafness and blindness.

Isoniazid by the intrathecal route did not alter the clinical course of five patients; (cases 1, 6, 7, 11, 12). Two of these died and three showed improvement not related to this therapy. In the other seven its use coincided with the improvement noted.

No toxic effects due to intrathecal isoniazid were encountered even when 100 mg. was administered in one dose.

DISCUSSION

The results of therapy of tuberculous meningitis have improved considerably during recent years and specially during the last two years when isoniazid became available.

In spite of these great advances we are often faced with patients suffering from tuberculous meningitis in whom the usual modes of therapy using streptomycin by the intramuscular route, isoniazid orally, and PAS orally fail to produce the desired results.

For this reason the administration of isoniazid into the subarachnoid space has been attempted hoping that the superior results obtained with intrathecal streptomycin, before isoniazid was available, may be reproduced or improved upon by using intrathecal isoniazid.

The earliest reports of isoniazid by the intrathecal route came from Italy[2] where the drug was administered intrathecally to dogs and was found to be non toxic; human experiments followed immediately: a slight increase in proteins and cells in the spinal fluid was noted in these cases. There were no serious toxic reactions and no neurological sequelae following the administration of isoniazid intrathecally.

Shortly afterwards, a report from Spain[5] indicated that isoniazid when used by the intrathecal route in doses of 20 to 30 mg. "appeared to be a good substitute for streptomycin intrathecally". In a series of 82 cases no toxic manifestations were observed with isoniazid. In this report it is not possible to ascertain whether the good results reported could just as well have been obtained with the oral isoniazid which was being administered at the same time. The mortality rate was only 7.3% in patients treated for 15 days or longer. Obviously this follow up is too short.

Another report from Uruguay[6] emphasized the lack of toxicity of isoniazid administered by the intrathecal route. Doses up to 100 mg. were administered intrathecally without toxic manifest-

ations. Ten patients were treated with intrathecal isoniazid in addition to systemic therapy and 8 survived for periods of 1 to 8 months.

Another report from Germany[7] concerns 8 patients treated with massive doses of oral isoniazid — up to 1000 mg. in addition to intrathecal isoniazid 25 mg. daily for 2 to 3 weeks. All the patients survived; however, follow up is too short to make final conclusions. No toxic effects were noted except for a rise in cells in the cerebrospinal fluid.

In our group of cases certain facts stand out: (1) isoniazid is harmless when administered by the intrathecal route. It is probably beneficial in two ways: (a) increasing its concentration in the cerebrospinal fluid, places more of it in direct contact with the tuberculous exudate. (b) the daily drainage of cerebrospinal fluid decreases the intracranial hypertension; (2) The fact that improvement in patients whose course was stationary or downhill coincided with the administration of intrathecal isoniazid suggests that this form of therapy should be attempted in all such cases. The main argument against this contention is that isoniazid passes easily through the blood cerebrospinal fluid barrier. Even in cases of spinal or basal block, isoniazid may be detected in adequate concentrations in the various portions of the subarachnoid space. This is shown in table I of patient G. S., a 9 month infant who had spinal block due to tuberculous meningitis, on whom we have reported previously.[8] Concentrations of 1 microgram per ml. were obtained in all portions of the subarachnoid space. A concentration of .034 micrograms/ml. is considered adequate to inhibit growth of the H. 37 RV strain. In a report from Spain[9] a thorough investigation of the blood and cerebrospinal fluid levels of isoniazid was made in 15 cases. In two patients values of 0 were obtained despite doses of 500 mg. and 900 mg. of isoniazid given orally to 56 kg. individuals. In all other cases adequate levels were obtained. The authors conclude that intrathecal isoniazid is not necessary, ever, and discard these results of 0. They mention that Ramos and co-workers recommend intrathecal isoniazid in seriously ill or stationary cases. We have done this in several cases with the results that are being reported in this article. It might be postulated that isoniazid sometimes fails to appear in the cerebrospinal fluid in the same manner that a decrease in the passage of glucose from the blood into the cerebrospinal fluid occurs[10] in most cases of tuberculous meningitis. Such patients would certainly benefit from intrathecal therapy and should not be penalized by an inflexible no-intrathecal-therapy rule. Ideally, routine cerebrospinal fluid levels of isoniazid should be made in all cases in order to determine the need for intrathecal therapy.

A simple method to determine isoniazid values would therefore be highly desirable.

TABLE I

Region	Time after oral INH 4 mg/kg.	CSF values of INH in Mcgm/ml.
Ventricular	9 hrs.	1.39
Cisternal	9 hrs.	1.23
Lumbar	9 hrs.	1.35

ISONIAZID LEVELS in the ventricular, cisternal and lumbar regions in a patient with tuberculous meningitis, spinal block and hydrocephalus.

SUMMARY AND CONCLUSIONS

1. Isoniazid has been administered by the intrathecal route to twelve patients with tuberculous meningitis.
2. It was well tolerated by all patients and appeared to be of benefit in 7 cases.
3. More cases and a controlled study are needed in order to confirm these preliminary findings.

SUMARIO Y CONCLUSIONES

1. Se usó isoniacida por vía intrarraquídea en 12 enfermos con meningitis tuberculosa.
2. Fué bien tolerada y ha dejado una impresión favorable en sus resultados clínicos en 7 enfermos.
3. Un aporte casuístico de mayor volumen (con casos testigos) es necesario para confirmar nuestras impresiones iniciales.

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NON-TRAUMATIC ACUTE PERFORATION OF THE COMMON HEPATIC AND COMMON BILE DUCTS

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Acute perforation of the common hepatic and common bile ducts is such a rare occurrence that few practitioners ever have the experience of dealing with one of these cases. On the other hand the seriousness of this condition is such, the mortality so high, that we believe a proper review of the literature up to the present should be made, adding two new cases that have come to our attention, with the view of searching for guides to the earliest possible recognition of this complication and the best way to treat it.

Although it is not the object of our discussion, we desire to mention that non surgical traumatic rupture of the common hepatic and common bile ducts has also been encountered rarely, mainly in children and young adults and that this catastrophe if duly recognized has been handled more successfully since the diagnosis is more evident and barring additional serious trauma, it does not present the complicated biliary pathology encountered in the non-traumatic cases.

Since the clinical features are about the same whether the rupture occurs in the common bile duct, in the common hepatic or in the stump of the cystic duct, we have decided to consider all of them together. Some of the reported cases do not give the exact location of the rupture; in others the diagnosis is presumptive. Moreover, the frequent anatomical variations in the point of junction of the cystic and common hepatic ducts and the fact that the cystic duct may even join the duodenum separately, the distortions brought about by previous surgical interventions and inflammatory reactions add to the difficulties and confusion in localizing the exact point of perforation.

In spite of the rarity of this condition, the literature on the subject is fairly voluminous and time limitations do not permit us to enter into it in detail. We just want to mention that already in 1799 Wainwright reported a case of rupture of the hepatic duct and in 1806 Fizeau published a case of rupture of the choledochus with bile peritonitis. These were followed by Freelands paper in 1882 and various other contributions down to the present including the often quoted report of McWilliams (1912).

A brief resume of two cases coming under our observation follows:

Case No. I— M. V. M., female, 8½ months pregnant admitted to the O. B. Department of San Juan City Hospital, March 20, 1953, because of a dull aching pain in epigastrium and RUQ of three days duration. Constipated for four days. Since eight years old had suffered belching and bloating with RUQ discomfort after fatty food ingestion. Had painless icterus after delivery of second child 5 years previously and had suffered RUQ pain on the sixth month of the same pregnancy. Temp. was 100.4°F. There was marked generalized tenderness, more noticeable in the RUQ and rebound tenderness referred to the latter region. W.B.C. count was 13,650 of which 95% were neutrophiles. Urine gave a positive reaction for urobilinogen. A diagnosis of acute cholecystitis was made and since the pain appeared to subside, was treated with antibiotics and Wangensteen suction. On March 23 delivered spontaneously a living baby, but two days later symptoms recurred with marked toxemia and a paralytic ileus. On operation the peritoneal cavity was discovered to contain about 3 liters of deeply bile stained fluid and thin fibrinopurulent adhesions. There was an anterior perforation about ¼ inch in diameter just above the cystic duct. A T-tube was inserted through the perforation and a drain was left in Morrison's pouch. After an initial stormy convalescence the patient recovered and on the 16th. operative day she was discharged after a cholangiography showed a patent common duct. Post operative follow up two weeks later showed the patient had continued well. This patient was operated upon by Dr. Gumersindo Blanco who kindly gave us permission to use it.

Case No. II. D. R. B., female, white, 80-years-old, admitted to Hospital Mimiya, September 11, 1954, because of RUQ pain. Seven and a half years previously an appendectomy was done in Miami for acute appendicitis and 1½ years later a piece meal cholecystectomy had to be done for a very severe acute gangrenous cholecystitis with cholelithiasis. Remained well till about 1½ years previous to this last episode when she began to experience frequent attacks of high fever with moderate leucocytosis and hyperbilirubinemia lasting one to three days. A diagnosis of choledocholithiasis was made and operation was repeatedly advised which she always refused. Finally, after a period of 9 months free from these attacks, she suddenly developed a severe persistent pain in the RUQ with fever up to 103°F, leucocytosis of 15,200 with 94% neutrophiles, Sr. bilirubin of 4.25 mgm % and Sr. amylase 35 units. She was hospitalized on the third day and with i. v. fluids, vitamins, antibiotics and sedation, improved considerably the first few hours but on the second day developed a LLQ pain

which spread throughout the abdomen with distention. Two days after admission the serum bilirubin rose to 8.65 mg.%, fell to 5.42 mg.% and later rose again to 6.9 mg. A preoperative diagnosis of common duct rupture with bile peritonitis was made and operation was decided upon. She was operated by one of us, Dr. A. S. Casanova Diaz. On entering the abdomen a large amount of bile was found located mainly in the left subphrenic space, left lumbar gutter, pelvis and right anterior subphrenic space. The right gutter contained no bile due to previous adhesions. Due to the very poor condition of the patient further exploration was deemed unadvisable and a sump drain was placed in the area of the foramen of Winslow and another in the left gutter and the abdomen was closed.

Following surgery she improved greatly but leucocytosis and hyperbilirubinemia continued. A spiking fever developed which by October 4 had reached 103ºF. It was our feeling that she had an acute suppurative cholangitis secondary to obstruction and since she was losing ground a second operation was decided upon. Marked omental and peritoneal adhesions were encountered forming a solid wall about the liver. After tedious separation the common bile duct was found of normal size though somewhat thickened. The site of previous perforation appeared to have been in the region of the porta hepatis where a bile pigment scar was found on the wall of the choledochus. A few small black soft pigment granules were present in the common duct with some mud. Small dilators passed into the duodenum with some difficulty; larger ones could not be passed. A T-tube was placed in the common duct and brought out through a stab wound. Following surgery Hanger test was 3 plus. Despite blood transfusions, intravenous fluids, antibiotics and other vigorous supportive measures, the patient's condition deteriorated rapidly and eleven days after the second operation she passed away. Permission for autopsy could not be obtained.

On the basis of our experience and the literature we have reviewed we have classified acute perforations of the common bile ducts, hepatic duct and cystic stump as follows:

A. Traumatic

B. Non traumatic

1. Without previous biliary surgery.
2. After cholecystectomy
3. After cholecystectomy with choledochostomy.

The principal etiological factors may be enumerated as follows:

A. Obstruction

1. Stone, biliary mud or both.
2. Fibrotic stenosis of sphincter of Oddi.
3. Spasm of sphincter of Oddi.
4. Blockage by mucus plugs, tumors or parasites.

B. Weakening of wall of common duct.

1. Previous choledochotomy.
2. Compensatory dilatation after cholecystectomy.
3. Dilatation secondary to obstruction at lower end.
4. Ulceration from pressure by calculus.
5. Calculus impacted in duct wall.
6. Thrombosis of wall vessels with localized gangrene.
7. Repeated attacks of choledochitis.
8. Necrotizing action of pancreatic reflux.

Perhaps as a plausible explanation of the rupture of the common duct in some cases we want to call attention to Burden's paper on "Histologic and Pathologic Anatomy of Hepatic, Cystic and Common Bile Ducts." He showed that the extrahepatic bile ducts contain numerous glands in their walls which extend almost to the peritoneal coat and normally are filled with mucus secretion. In a dilated diseased duct an infection of one of these glands or a calculus formation may be the cause of weakening and rupture of the duct.

The typical case is usually a female above forty with a long history of frequent biliary colics, often accompanied by jaundice, fever, nausea and vomiting. In the large majority of instances there is a history of previous biliary surgery with cholecystectomy and frequently also choledochostomy. The time interval for the subsequent perforation has varied from fifty-eight days as in one of the cases reported in 1935 by Wolfson and Levine to six years as in one of our cases which to our present knowledge is the longest interval on record.

After a variable asymptomatic period there is an acute recurrence of pain which suddenly become very severe, at times excruciating, referred often to the back or even to the lower abdomen, depending on the amount of bile escaping into the peritoneal cavity. The patient as a rule appears severely ill and the pain often persists even in spite of fairly large doses of narcotics. Nausea and vomiting as in the typical choledochal syndrome is al-

most always present. Jaundice is often evident and even in spite of rupture may continue to increase for some time as it happened in one of our cases in which the serum bilirubin went up from 4.25 mgm. to 8.65 mgm. per 100 cc. of blood.

After the frequent initial shock the temperature tends to rise to variable levels and the pulse which may be weak and rapid in the markedly depressed patient may be relatively slow when jaundice is deeper.

The abdomen is tender, sometimes distended, with fluctuation, with some resistance to palpation, occasionally a board-like rigidity. In cases in which the escape of bile is localized these findings may be limited. Generally, one will find a peritonitis of the RUQ which may spread to the rest of the abdomen. However this does not always hold, for depending on the spread of bile, symptoms may initially be more marked in the right or even LLQ as it happened in one of our cases where the escaping bile flowed down the left gutter, the right gutter being blocked by adhesions from a previous appendectomy and an antecedent cholecystectomy.

At this stage the patient appears very toxic, high leucocytosis is usually present. A very typical finding is the absolute absence of peristalsis after a short initial period.

Differential diagnosis is mainly from:

A. Acute biliary colic. The case is one who has usually been having similar colics and both he and the attending physician feel inclined at first to diagnose it as one of the usual attacks which will respond to the measures formerly employed.

B. Acute pancreatitis. Serum amylase determination will help to exclude this possibility. A normal figure if the test is made at the onset rules out pancreatitis except in the occasional case of a markedly necrotic process in which other clinical findings would help to make the proper diagnosis. Serum amylase may be elevated in any type of peritonitis but the figures are not as high as in a comparable case of pancreatitis.

C. Perforated peptic ulcer is usually accompanied by a preceding history of peptic ulcer disease. The abdominal board-like rigidity is more usually present and a simple X-Ray plate will often show a gas bubble under the diaphragm.

D. Acute perforated appendicitis has been diagnosed preoperatively in a few of the reported cases. Only by a proper analysis of the history and physical findings, the differential diagnosis can be made in a few of those cases in which the bile escaping into the RLQ provokes an inflammatory reaction in this region.

Moreover in all doubtful cases we must recur to abdominal aspiration for the finding of typical bile or bile salts on one hand, or pancreatic enzymes on the other are helpful guides to achieve a correct diagnosis.

TREATMENT

Treatment of acute perforation of the choledochus is surgical. The free bile should be removed by aspiration, and the common duct explored as soon as the diagnosis of perforation is arrived at. The only acceptable delay should be that which is necessary to prepare the patient for the contemplated surgery.

Exploration of the common duct may not be advisable under certain circumstances. If the patient is in very poor physical condition and exposure of the common duct is rendered difficult because of old and recent adhesions, it would be wiser to limit the surgical procedure to simple evacuation of free and loculated bile, and insertion of a drain in the region of the duct. Occasionally, it may also be advisable to drain other areas of loculation.

If trainage alone is done at the 1st intervention, exploration of the common duct should be carried out as soon as the condition of the patient warrants it.

At the exploration of the common duct, all etiological factors for the perforation should be looked for and eradicated, if possible. Stones should be removed, strictures corrected, and a spastic sphincter dilated (or divided transduodenally). However, if the conditions of the patient during the operation becomes too poor, it is advisable to back out, leaving a T- tube in place. The remaining defects can be corrected at another operation later on after the patient is prepared for it. It is much better to have a patient with an obstructed common duct, draining bile through a T- tube, than a dead patient with a patent choledochus. The former you can correct at a later date; the latter you can only regret.

In some cases, after the perforation has occurred, the choledochus remains patent and there is no residual major obstruction. The perforation may then seal off spontaneously, and the continuity of the choledochus is thus restored. No external biliary fistula develops (or if present for a time, closes), and jaundice subsides completely or may never be present.

If we could determine beforehand which are these fortunate cases, then we could save them the risk and discomforts of a serious major surgical procedure. We may have the solution to this problem in the visualization of the hepatic and common ducts with the recently developed radio-opaque substances which can be administered intravenously. Then we could forego the common

duct exploration in any patient in whom the bile ducts are shown to be free of stones and strictures and the flow of the radio-opaque substance into the duodenum is free.

Antibiotics definitely play an important role in the management of these cases. Bile peritonitis at first is purely chemical, but it does not take long to get contaminated, and we can prevent or control this by the adequate use of the broad spectrum antibiotics. Furthermore, the pulmonary complications which are so frequent and dangerous in this type of patient are, as a rule, also controlled satisfactorily with the antibiotics.

Finally, we want to give credit to the advances in anesthesia in the successful management of these cases. As a rule, they are older individuals who do poorly if hypoxia and hypotension are not prevented or adequately controlled.

CONCLUSIONS

1. Two cases of non-traumatic perforation of the common bile duct have been presented.

2. The longest interval between previous biliary surgery and spontaneous perforation has been recorded.

3. The fact that jaundice may continue to increase in spite of perforation has been noted.

4. Considering that perforation of the choledochus and other wellknown complications may result from coledocholithiasis, the danger of procrastination in this condition is apparent.

5. Factors other than common stone which may be responsible for perforation have been discussed.

6. The importance of surgical intervention at the earliest possible moment is apparent.

7. The use of diagnostic abdominal needle aspiration has been mentioned.

8. Differential diagnosis has been discussed.

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DIVISIÓN DEL CONDUCTO ARTERIOSO*

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Munro[1] en 1907 fué el primero en sugerir la ligadura del conducto arterioso, también llamado canal arterial. El primer intento de operación lo hicieron Graybiel y Strieder[2] en 1938. Gross y Hubbard[3] informaron la primera ligadura con éxito en 1939. Shapiro y Keys[4] coleccionaron 107 casos de conductos no infectados. La operación tuvo éxito en 81 de ellos y nueve murieron durante la intervención o poco tiempo después. Desde entonces con los adelantos en técnicas y experiencia la mortalidad ha disminuído enormemente. Gross[5] en 1952 informó 525 casos operados con una mortalidad total de 2%. La mortalidad de los casos que sólo tenían síntomas mínimos antes de la operación fué de solamente 0.5%. Potts[6] en una serie de 214 casos no tuvo ninguna muerte.

Shapiro y Johnson[7] en 1947 analizaron 525 casos no infectados los cuales fueron tratados por el método de ligadura por 46 cirujanos distintos encontrando una incidencia de recanalización de 8.7%. Por ese motivo consideraron la ligadura del canal como anticuada y sólo la recomendaron en casos donde es imposible hacer la división. El fracaso de la ligadura lo atribuyeron a lo difícil en saber cuál es la tensión óptima que se debe aplicar a las ligaduras; si muy poca, el canal no se ocluye totalmente; si excesiva, la ligadura corta a través.

Además, no es deseable el dejar grandes cantidades de sutura gruesa no absorbible cerca del bronquio y los grandes vasos. Gross[5] también prefiere la división del conducto teniendo en mente que es un principio fundamental de cirugía vascular el dividir una arteria grande para poderla ocluir.

Pronóstico en los casos no operados

Cuando un canal arterial permanece abierto el individuo tiene una comunicación que es parecida a una fístula arteriovenosa. La comunicación puede tolerarse bien siempre y cuando el individuo se mantenga libre de infecciones y la comunicación sea suficientemente pequeña para no recargar mucho el trabajo del corazón. Con estas circunstancias favorables el individuo puede tener una larga sobrevida con poca o ninguna incapacidad. Sin embargo estas condiciones ideales sólo se encuentran en raras ocasiones.

Hay ciertos peligros en los pacientes con un canal abierto:

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* Trabajo presentado en la Asamblea Médica del Distrito Sur el 14 de agosto de 1954 en Ponce, Puerto Rico.

1. A través del canal puede pasar tanta sangre que haga disminuír la circulación periférica y produzca en el individuo un crecimiento retardado.
2. El corazón puede aumentar su débito tratando de mantener la circulación periférica a un nivel satisfactorio y entonces se recarga grandemente.
3. La infección bacteriana puede asentarse sobre la anormalidad siendo el organismo agresor el streptococcus viridans. La incidencia de esto es de un 25%.[6]
4. Puede ocurrir una dilatación aneurísmica y rotura del canal.

Mientras que la presencia del canal en el niño aparenta ser un problema inocuo las observaciones a largo plazo han revelado con gran frecuencia un desenlace serio. En la serie de Abott[8] la quinta parte de los pacientes murieron en la infancia. La edad promedio del resto al morir fué de veinticuatro años. Bullock, Jones y Dolley[9] coleccionaron 30 casos de más de tres años. El 50% murió antes de los 30 y el 71% antes de los 40. En el 87% la causa de muerte fué de origen cardíaca. Chapiro y Keys[4] encontraron que la edad promedio al morir era de 30 años en los varones y 35 en las mujeres. El 40% moría de endocarditis bacteriana y el 30% de insuficiencia cardíaca.

Selección de Pacientes para Operación

Es generalmente aceptado que se debe operar a todos los niños afectados que tienen un desarrollo físico retardado.[6] La insuficiencia cardíaca es también una indicación absoluta. También es aceptable la operación en todos los individuos de 30 a 50 años, que aunque no muestren evidencias marcadas de insuficiencia cardíaca se encuentren fatigosos y tengan que reducir su actividad física.

Los pacientes que tienen endocarditis deben ser operados. Hay que recordar que cuando la infección cede, muchas veces ellos permanecen como inválidos cardíacos debido a la cicatrización que ocurre en el miocardio. En esos casos es imperativo el reducir la carga mecánica sobre el corazón por medio del cierre del canal arterial. Los pacientes infectados deben ser sometidos a tratamiento intenso con antibióticos por un período de tiempo adecuado; de ser imposible esterilizar la sangre por sólo este método, debe hacerse la operación en el período activo. En 1940 Touroff y Vessel[10] demostraron de una forma dramática que esta infección puede curarse en un 75% de los casos con la oclusión del conducto. Si es posible esterilizar la sangre con drogas solamente, la operación puede posponerse.

Después de considerar las categorías anteriores todavía queda un gran número de pacientes, particularmente en la infancia,

que no tienen sintomatologia alguna y que frecuentemente nos colocan en la obligación de decidir si la operación debe o no hacerse. La contestación a esto depende de la capacidad del cirujano de llevar a cabo el cierre completo del ductus con una mortalidad mínima. ¿Por qué correr el riesgo de la insuficiencia cardíaca o de la endocarditis bacteriana cuando pueden prevenirse ambas con la operación? Además hay que tener en cuenta que las operaciones del canal arterial se hacen con mayor facilidad en los más jóvenes. El adulto presenta más dificultades porque los vasos son más rígidos y la reserva cardíaca menor.

TECNICA DE LA OPERACION

Con el paciente en decúbito lateral derecho hacemos una toracotomía posterolateral izquierda. Entramos al hemitórax izquierdo a través del cuarto espacio intercostal sin seccionar costilla alguna. Retraemos el pulmón hacia abajo y dividimos la pleura mediastínica longitudinalmente entre el frénico y el vago. Procedemos a hacer la disección en esta región logrando identificar el canal arterial. Limpiamos la extremidad aórtica del canal. La extremidad pulmonar del canal la disecamos después de abrir el pericardio, lo cual hacemos rutinariamente. De este modo podemos limpiar el conducto en su totalidad y separar el recurrente sin lesionarlo. Colocamos entonces dos pinzas de ductus de Potts[11] o las modificadas por Glover, y entonces suturamos ambos muñones con seda arterial 4-0. Dejamos el pericardio parcialmente abierto y cerramos el mediastino. Cerramos el tórax con drenaje bajo agua.

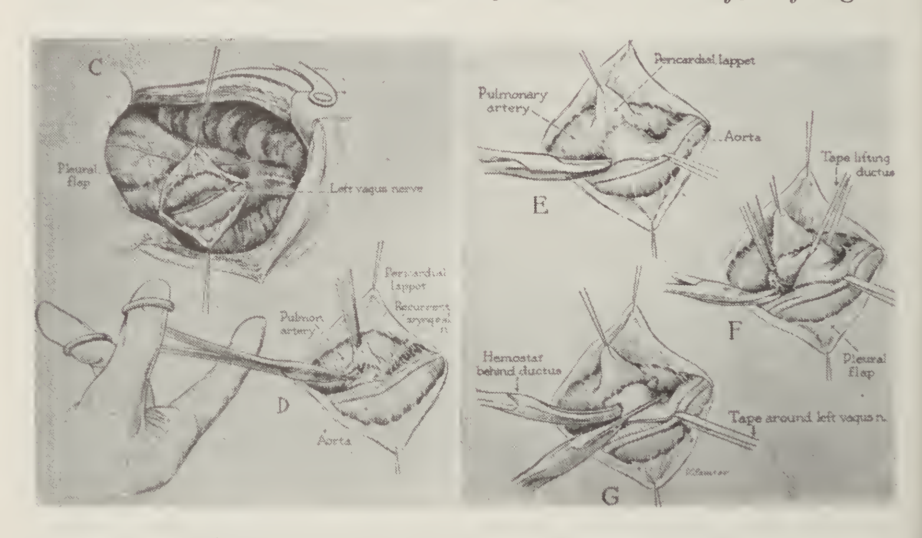


Fig. 1 Fig. 2

Fig. 1. Se ha abierto la pleura longitudinalmente entre el nervio frénico y el vago.

Fig. 2. Se ha hecho la disección del canal arterial. Se ha pasado una cinta alrededor y se ha colocado una pinza.

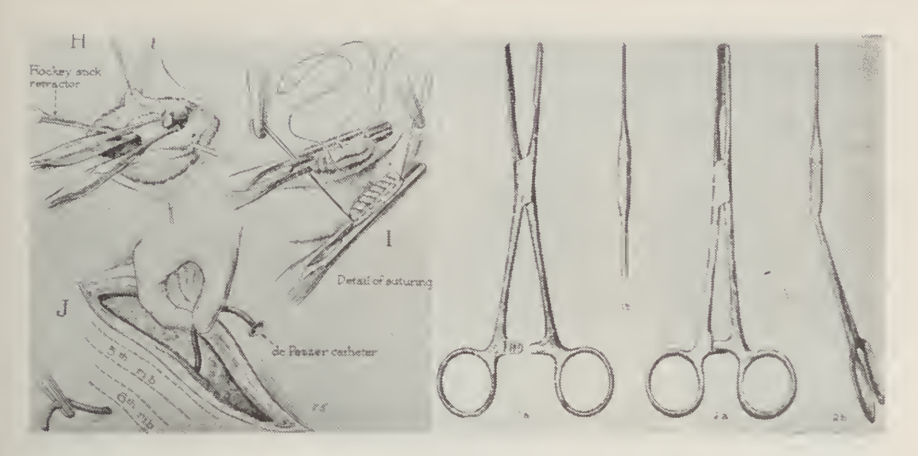


Fig. 3 Fig. 4

Fig. 3. Se han colocado dos pinzas de Potts, se ha dividido el conducto y se han suturado los muñones.

Fig. 4. Vistas de frente y de lado de las pinzas de Potts.

PRESENTACION DE CASOS

Caso 1 - J.L.R. Hombre de color de 35 años, admitido al Hospital de Damas el 10 de agosto de 1953. Dió la historia de haber tenido dispnea cuando niño. Como adulto siempre se había sentido bien. El examen físico reveló un frémito sobre el precordio. Había un soplo continuo del tipo llamado de maquinaria que podía oírse sobre todo el lado izquierdo y con más intensidad en el segundo espacio intercostal izquierdo. El soplo se transmitía hacia arriba y a la izquierda. La presión arterial era de 140/80. En las radiografías aparecía un marcado aumento en el cono pulmonar. La radioscopía mostraba la típica danza hiliar. Hicimos el diagnóstico de conducto arterioso. El 17 de agosto de 1953 hicimos una toracotomía izquierda y confirmamos el diagnóstico. Hicimos una división y sutura del conducto. El paciente toleró la intervención bien. El post-operatorio se complicó con abundantes secreciones bronquiales, atelectasia de lóbulo medio derecho y un pequeño derrame pleural izquierdo. Estas complicaciones se resolvieron sin consecuencias serias, el paciente recobró y fué dado de alta del hospital el 8 de septiembre de 1953.

Caso 2. — A. S. Hombre de 21 años fué admitido a la Clínica Dr. Pilá el 1ro. de diciembre de 1953 para ser sometido a una operación para glaucoma crónico. En el examen rutinario se encontró el frémito y el soplo característicos del canal arterial. La presión arterial era de 128/68. Las radiografías no mostraron la silueta cardíaca típica del conducto. El electrocardiograma mostró una leve hipertrofia del ventrículo izquierdo. El 3 de diciembre de 1953 hicimos la toracotomía y encontramos un conducto de 1.5

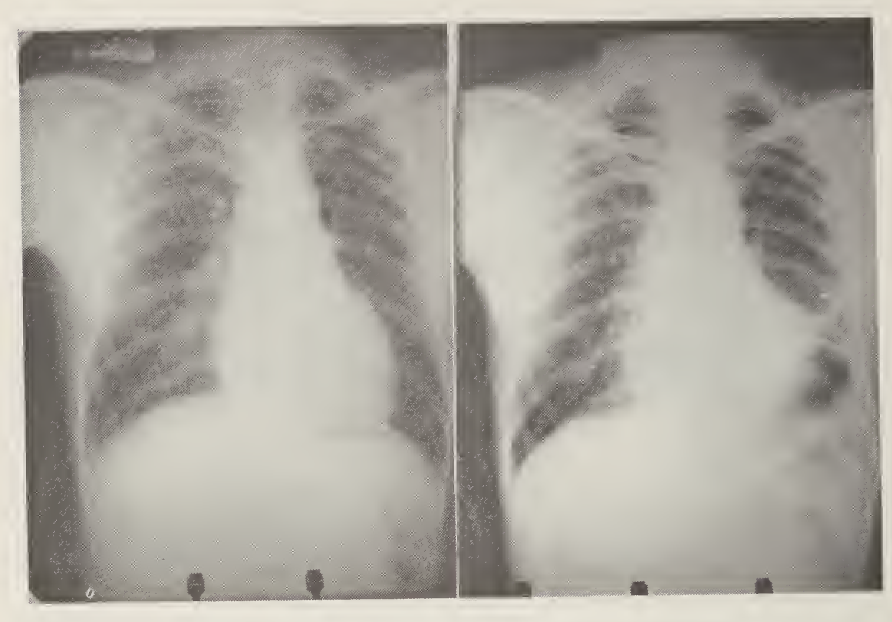


Fig. 5 Fig. 6

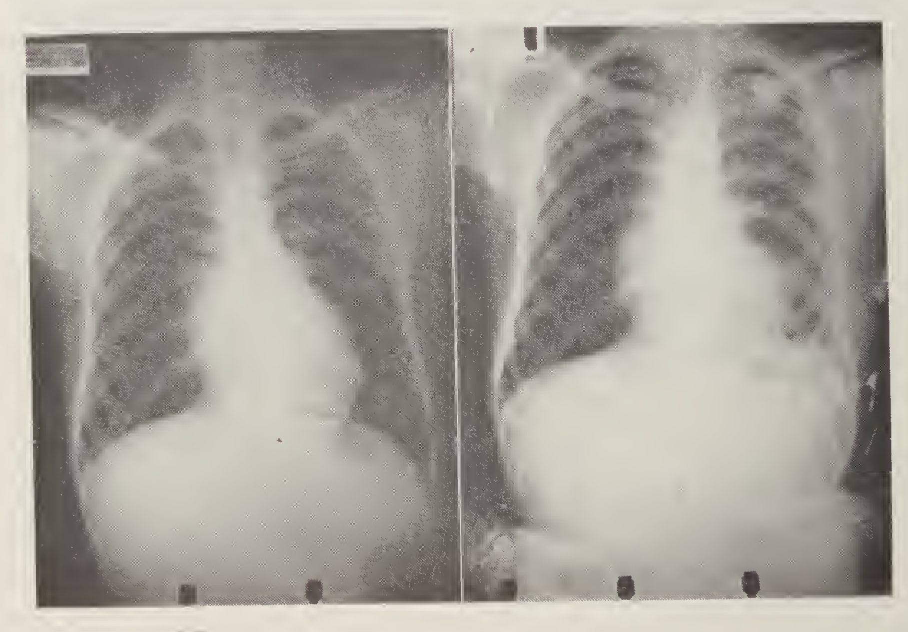
Fig. 5. Radiografía del caso núm. 1 preoperatoria demuestra un leve aumento del cono pulmonar.

Fig. 6. Vista postoperatoria del mismo después de dividir el conducto arterioso.

Fig. 7. Vista preoperatoria del caso 2 demuestra un leve aumento del cono pulmonar.

Fig. 8. Radiografía del mismo postoperatoria después de división y sutura del conducto.

Fig 7 Fig. 8



cm. de diámetro. Fué tratado éste también por división y sutura. El paciente estuvo en perfectas condiciones durante la operación y tuvo un postoperatorio sin complicaciones. Fué dado de alta del hospital el 14 de diciembre de 1953.

RESUMEN Y CONCLUSIONES

Hemos hecho una breve historia de la operación, hemos discutido el pronóstico en los casos no operados y también la selección de pacientes para la operación.

Presentamos nuestra experiencia y la técnica usada. Presentamos nuestras excusas por la casuística limitada pero creemos que es de valor porque son las primeras experiencias en nuestro medio de división del canal.

Creemos que la corrección operatoria del conducto arterioso no complicado ha llegado a ser una operación extremadamente satisfactoria. Podemos con toda honradez ofrecer esta operación con una oportunidad excelente de sobrevivir a ella y de tener un corazón normal después.

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GUILLAIN-BARRE SYNDROME

PRESENT CONCEPTS AND REPORT OF 14 CASES

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Since the original description of Guillain, Barré and Strohl[1] in 1916, the clinical entity characterized by an acute polyradiculoneuritis and albuminocytologic dissociation in the cerebrospinal fluid has been extensively studied. This syndrome has been variously described as infectious neuronitis, acute polyneuritis with facial diplegia, myeloradiculitis, polyradiculitis, neuritis with albuminocytologic dissociation, and Guillain-Barré syndrome.

The ever increasing importance of diagnosing this disease in daily practice has inspired this communication. Many of our cases were thought to be poliomyelitis by the referring physicians; thus, it is of paramount value to examine the clinical manifestations of this syndrome so that its correct diagnosis can be made more frequently. Guillain-Barré syndrome will not be missed if it is considered whenever an ascending, progressive, symmetrical paralysis with preponderance of motor involvement over sensory disturbances, with or without facial diplegia, is encountered. A considerable number of cases present themselves with simultaneous involvement of the four extremities, at times with radicular pains not to be confused with polyneuritis. An associated hyperalbuminosis in the spinal fluid without a corresponding increase in cells will establish the diagnosis.

MATERIAL

Fourteen case-records were obtained for statistical studies from the files of the Bayamón District Hospital, covering the period from January, 1951 to July, 1954.

RESULTS

Age: Of the 14 patients, four were children; ten (71%) were adults. The youngest was 2 years old, and the oldest 78. The average age in this group was 28.3 years.

Sex: Eight cases in this group were males and six were females. Thus, no sex predominance was evident.

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Duration of illness: The average duration of the acute stage of the disease was approximately 8 weeks, the longest being 10 weeks and the shortest 3 weeks. This determination includes the thirteen cases that recovered. The other one, the only fatality, died 22 days after the onset of symptoms.

Physical Signs: Pertinent findings were limited to the neurologic examination. All the cases showed involvement of the motor system. In seven (50%) patients, weakness or flaccid paralysis of the four extremities was found, while in the other seven, involvement of the motor system was limited to the lower extremities. Neurologic findings included flaccid paraplegia or quadriparesis, absence of deep tendon reflexes, and in one case, atrophy of the leg muscles. Facial diplegia was found in four (28%) of the fourteen patients.

Sensory changes were usually mild in nature, and were found in seven (50%) patients. These changes were usually spotty areas of hypesthesia in the lower extremities, although in three, there were sensory levels to the lower thoracic and upper lumbar segments.

Fever: Three cases had low grade fever.

Sphincter disturbances: Seven (50%) of the fourteen patients had sphincter disturbances, evidenced by urinary retention or urinary incontinence. Two had an associated bowel incontinence.

Laboratory studies: The only important positive laboratory findings were those related to the spinal fluid study. In all, there was an albuminocytologic dissociation. The highest cell count was 5 per cu. mm. while the protein determination varied from 58 to 432 mg%. The Pandy's test was positive in all cases. The fatal case had no cells in his cerebrospinal fluid, 320 mg% of protein, 63 mg% of glucose and three plus Pandy's test.

Treatment: Management in all cases consisted of bed rest, and tidal drainage whenever urinary retention was present. As far as drug therapy, vitamins B_1 and B_{12} were given in several cases, and three received cortisone. The duration of illness in these three cases was 25, 48 and 28 days, the average being 34 days, much less than the average duration of disease in the other cases. However, the patients treated with cortisone were too few to warrant conclusions as to the effectiveness of cortisone in Guillain-Barré syndrome.

Mortality: One case died in this series, for a mortality of 7.1%. Death was due to respiratory failure in an ascending type of paralysis involving the respiratory center. The patient was a 34 year old man admitted with the history of numbness of the lower extremities of 18 days duration, progressing to quadriplegia and absence of deep tendon reflexes with no sensory impairment.

The patient died four days after admission, developing respiratory distress and terminal cyanosis.

DISCUSSION

In their original description of Guillain-Barré syndrome,[1] the authors stressed a triad found in this disease: albuminocytologic dissociation in the cerebrospinal fluid; preponderance of motor weakness over sensory disturbances; and, favorable prognosis, usually with complete recovery. The latter has been found not to be invariably true, as fatalities may occur. Fox and O'Connor[2] reported a mortality of 20% in 126 cases; Forster, Brown and Merrit[3] one of 42% in 26 cases, and Roseman and Aring[4] one of 18.8% in 16 cases. In 1949 Haymaker and Kernohan[5] in an excellent clinicopathologic report, concluded that no essential difference existed between Guillain-Barré syndrome and Landry's ascending paralysis, and they suggested the name Landry-Guillain-Barré syndrome to emphasize that the prognosis is often grave.

As far as incidence, the 14 cases reported in this communication, seen in a period of three and a half years (January 1951-June 1954), compare favorably with the frequency in which this clinical entity was found in the Henry Ford Hospital, Detroit, Michigan. Raftery et al[6] stated that 34 cases were encountered in a period of ten years in that institution.

The clinical course of the cases of this series paralleled that of other reports.[7] The disease was present in some children, but more often affected adults, with no difference in incidence as far as sex. A rapidly progressive motor paralysis of the extremities was the rule, the lower extremities being affected more severely than the upper extremities. In 28% of our cases, paralysis of the seventh cranial nerve in one or both sides was found. This is in accord with other authors[8] who report an incidence of facial diplegia of approximately 35%. Sensory symptoms although as a rule not as marked as the motor ones, formed part of the clinical picture in many instances. Fifty per cent of our cases had vesical and/or anal sphincter difficulties. The course was usually afebrile and the typical albuminocytologic dissociation was found in the spinal fluid.

The cause of Guillain-Barré syndrome has been generally considered to be a toxic or infectious agent, either bacterial or viral. Attempts to identify the causative agent have been unsuccessful. The condition generally develops a few days or weeks after a mild infection, often a common cold. Several reports have appeared of cases of Guillain-Barré syndrome associated with infectious mononucleosis[6,9] and infectious hepatitis,[10]

Treatment, depending on varying concepts, has changed considerably, and since the condition is usually self-limited, evaluation of therapy is difficult. Various members of the Vitamin B complex have been administered; dimercaprol (BAL) has been used with success by others. More recently, corticotropin (ACTH) has been given a trial by several investigators.[11,12] They base their rationale for its use on the feeling that the disease might be due to a hypersensitivity of the nervous system to an allergen, the same as rheumatic fever is related to bacterial infection. Blood, Locke and Carabasi[11] and Essilier and Kop[12] have reported favorable response to ACTH in this syndrome, with relapses occurring on discontinuation of the drug and improvement on reinstitution of treatment with the hormone. As mentioned before, only three cases of our series received cortisone and in all the duration of illness was much shorter than in the rest of the group. It seems that ACTH or cortisone should be tried in severe cases where the prognosis is guarded. Results obtained by others with these hormones merit further study.

In the differential diagnosis of Guillain-Barré syndrome, it is important to consider conditions like polyneuritis due to diphtheria toxin, heavy metal poisoning, vitamin B defficiency, alcoholic neuritis, poliomyelitis, syphilis and spinal cord tumor. Diphtheria neuritis may be recognized when diphtheria throat lesions and positive cultures are present and syphilis by serologic tests in the blood and spinal fluid. A spinal cord tumor usually gives local signs and a careful manometric study will establish the diagnosis. Peripheral neuritis due to alcoholism and vitamin B defficiency usually yields more pain and no spinal fluid alterations. Heavy metal poisoning can usually be excluded by the history of exposure and the blood picture.

Guillain-Barré syndrome may often be mistaken with poliomyelitis. In the latter, cases occur usually in epidemics, the course is febrile and there may be meningeal irritation and severe spasm of muscles, not seen in Guillain-Barré syndrome. Poliomyelitis notoriously gives an asymmetrical type of paralysis, involving muscle groups such as the shoulder girdle, hip girdle and hamstrings. On the other hand, Guillain-Barré syndrome is distinguishby a symmetrical distribution of paralysis. In the preparalytic stage of poliomyelitis, there is pleocytosis of the spinal fluid, consisting early of polymorphonuclear leukocytes and later of lymphocytes. If the fluid is examined several weeks after the onset of poliomyelitis, a normal cell count and a high protein content are usually found in both conditions.

SUMMARY

1—Guillain-Barré syndrome is a clinical entity characterized by a rapidly progressive flaccid motor paralysis of the extremities, frequently with mild to moderate sensory changes. Facial diplegia is found in about 30% of the cases, and sphincter disturbances in an appreciable number of patients. An albuminocytologic dissociation of the spinal fluid is found in all cases.

2—The course of the disease is usually benign, although deaths are not infrequent. In this series of 14 patients, one (7%) died from an ascending paralysis with respiratory failure.

3—The present concepts as to etiology and treatment have been discussed.

4—The differential diagnosis, with special references to poliomyelitis, has been stressed.

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VOCAL CORD PARALYSIS OF INTRATHORACIC ORIGIN

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The subject of vocal cord paralysis is of interest not only to the Otolaryngologist who is consulted because of hoarseness or dysphagia, but also of great interest to the Neurologist, the Internist and the Chest Surgeon because we are not dealing with a disease entity in itself, but with a sign of disease elsewhere, namely: within the cranium, neck, thorax or mediastinum.

In general there are three main groups of laryngeal paralysis:

1. Paralysis due to surgical trauma of the recurrent nerve.
2. Paralysis as a manifestation of a disease affecting the cerebro-spinal axis such as Multiple Sclerosis.
3. Associated laryngeal paralysis due to various combinations of lesions affecting the IX, X, XI, XII cranial nerves and at times other cranial nerves and the phrenic nerve.

Let us review the nerve supply of the larynx.

The somatic motor component of the Vagus nerve arises in the caudal part of the Nucleus Ambiguus at the lateral part of the Medulla. This area of the brain is supplied by the Posterior Inferior Cerebellar artery, which in turn is the largest branch of the Vertebral artery.

The impulses brought to the cells of origin of the Recurrent laryngeal nerve arise from both pre-central regions of the cerebral cortex.

This bilateral supra-nuclear innervation of the vocal cords was first demonstrated by Krause in 1884 and confirmed many times since.

The vagus nerve leaves the skull through the middle compartment of the jugular foramen included in the same sheath with the accessory nerve. In the neck the vagus pursues a vertical course lying in the Carotid sheath. The right Vagus crosses in front of the Subclavian artery and the recurrent laryngeal branch **hooks around** the artery to reach the groove between the trachea and esophagus.

The left Vagus also descends in front of the Subclavian artery enters the thorax behind the left Innominate vein. As the Vagus crosses over the arch of the aorta the recurrent laryngeal nerve arises on the left side of the arch and winds below the aorta at the point where the ligamentum arteriosum is attached and then ascends to the side of the trachea.

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Both recurrent laryngeal nerves ascend in the groove between the trachea and esophagus, pass under the lower border of the Inferior Pharyngeal constrictor muscle and enter the larynx behind the articulation of the inferior cornu of the thyroid cartilage with the cricoid. It supplies all the intrinsic muscle of the larynx except the Crico-thyroid muscle which is supplied by the external branch of the superior laryngeal nerve which arises from the Vagus near the base of the skull. This muscle is the main tensor of the vocal cord.

Any pathology affecting the Vagus nerve before the superior laryngeal nerve branches off will produce a complete paralysis of the true vocal cord. This will remain in the so called Cadaveric or intermediate position. The arytenoid cartilage will occupy a mid position and will be tilted forwards. The vocal cord will be relaxed due to lack of tensile stimulus thru the superior laryngeal nerve to the crico-thyroid muscle. The cord is flaccid and occupies a lower level than the unaffected cord. The tip of the vocal process of the arytenoid cartilage is directed inwards and appears as a definite prominence. There is a sufficient wide glottis for respiration but during phonation there is feebleness of the voice due to the air waste caused by the inability of the normal cord to close the glottis.

When the recurrent laryngeal nerve alone is affected then an incomplete paralysis will result. The vocal cord will remain at a median position, the tension of the cord will remain normal since the crico-thyroid muscle still is active. The voice will return to normal in 90% of the cases. In the other 10% the cord will assume a cadaveric position due to atrophy of disuse of the crico-thyroid muscle.

Paralysis of the left vocal cord is more common than the right even if intra-thoracic pathology is excluded. The ratio is of 3:1.

There are two syndromes seen in lesions at the infra-hyoid level of the neck and mediastinum:

1. **Gigonoux Syndrome** — due to lesions affecting the recurrent laryngeal nerve and also spinal part of the Accessory nerve. The symptoms here are: hoarseness, torticollis and drooping shoulder. The signs found are: unilateral paralysis of the vocal cord and of the Sternocleidomastoid and Trapezius muscles.

2. **Klinkert Syndrome** — due to lesions affecting the recurrent laryngeal and the Phrenic nerves.

The symptoms are: hoarseness and dyspnea. The signs found are: unilateral paralysis of the vocal cord and of the diaphragm.

The etiologic factors of laryngeal paralysis at the various levels where pathology is usually found are listed below:

1. **Bulbar Origin** (comprise 10% of all cases seen).

   a. Multiple Sclerosis
   b. Thrombosis of Posterior Cerebellar artery.
   c. Tuberculoma at the Nucleus Ambiguus (Medulla).
   d. Syphilitic Meningitis about the Pons.
   e. Bilateral Cortical lesions.
   f. Bulbar Poliomyelitis.
   g. Aneurysm of Cerebral arteries.
   h. Brain Tumors.
   i. Amyotrophic Lateral Sclerosis.
   j. Arterial Hypertension.

2. **Base of Skull:** (at or near the Jugular Foramen)

   a. Tumors of the Naso-pharynx.
   b. Chordomas (arise from remains of the Notochord.)
   c. Osteo-Petrosis (Marble bone disease.)
   d. Fractures and Bullet wounds at the base of the skull.
   e. Toxic Neuritis (chemical and bacterial)
   f. Viral infections.
   g. Peripheral Neuritis (avitaminosis B)

3. **Pathology at the lateral Pharyngeal Space:**

   a. Peri-tonsillar abscess (usually tuberculous)
   b. Retro-pharyngeal abscess (usually tuberculous)
   c. Tuberculous adenitis of the neck.

4. **Infra-Hyoid part of the Neck and Mediastinum:** usually affect the recurrent laryngeal nerves but the Phrenic and Accessory (spinal) nerves may be also involved.

   a. **Mechanical Factors:**

      1. Aneurysm of the Aorta.
      2. Cardiac enlargement due to various causes, such as: Congenital heart disease; Rheumatic heart disease with mitral stenosis and dilated left auricle.
      3. Pericarditis due to various causes.

   b. **Inflammatory Factors:**

      1. Tuberculous fibrosis of the lung apex.
      2. Tuberculous adenitis of neck.

   c. **Tumors:** main cause of unilateral laryngeal paralysis.

      1. Carcinoma of the lung.
      2. Carcinoma of the Esophagus - most common cause in this group.

3. Benign and Malingnant tumors of the Thyroid gland.
4. Mediastinal tumors either primary or metastatic.
5. Tumors of the Carotid Body.
6. Carcinoma of the Trachea.
7. Enlarged Thymus especially during irradiation.

d. **Trauma:**

1. **Surgical**

   a. Post-Thyroidectomy: main cause of bilateral paralysis. Immediate - due to severance or edema of the nerve. Delayed - due to scarring.
   b. Post Pneumonectomy.
   c. Following surgery of the cervical esophagus.

2. **Non-surgical:**

   a. Birth injury
   b. Gunshot or stab wounds.
   c. Fractured clavicle.
   d. Trauma to the neck.
   e. Following retro-grade bouginage for esophageal strictures.

Four cases of paralysis of the true vocal cord due to intrathoracic pathology follows.

Case No. 1

Is that of a white sixty-eight year-old man who was admitted to the medical service of the Río Piedras Municipal Hospital. The patient was referred for laryngeal examination because of hoarseness and dysphagia with a diagnosis of a mediastinal mass and possible carcinoma of the larynx. Laryngoscopy revealed a paralysis of the left true vocal cord in the median position. No other laryngeal or hypo-pharyngeal pathology was found. Chest plate done read as follows "there is noted an emphysematous lung structure with moderate fibrotic changes at the perihilar areas. The superior mediastinum is widened specially to the left. This may be due to enlarged nodes or a mediastinal tumor mass. The left leaf of the diaphragm is high in position which may be due to paralysis of the phrenic nerve. The hila areas are normally outlined. The heart and great vessels are within normal limits."

Clinically we have the syndrome of Klinkert due to a tumor mass in the superior mediastinum which infiltrated the left lung apex and involved the arch of the aorta as found at autopsy.

Case No. 2

This was an eighty-year-old white male referred to my office by his family physician because of hoarseness of three weeks duration following aspiration of a tablet which he eventually coughed up. Laryngoscopy revealed the left true vocal cord paralyzed in the median position. The pyriform sinuses and the rest of the larynx was entirely normal. An esophagram was done which was normal. Chest plate done at the same time revealed the following "there is a large area of mottled opacities with tendency to confluence in the left upper lung delimited inferiorly by the oblique fissure and corresponding to the general distribution of the posterior apical segment of the left upper lobe. This area arises in the hilum and suggests a combination of retained secretions, decreased aeration and possibly superimposed infection. The findings are most likely due to a bronchogenic neoplasm."

The patient died within two months of Carcinoma of the lungs.

Case No. 3

This is a fifty-six-year-old white female referred to the Ear, Nose and Throat clinic of the San Juan City hospital because of hoarseness of 8 months duration. Indirect laryngoscopy revealed the left true vocal cord to be paralyzed in the median position. The rest of the larynx was found to be normal. Chest plate was ordered and reported as follows "there is moderate retraction of the left lung with fibrocalcific disease of the upper lobe and probable cavitation of the apex by tuberculosis. There is diffuse infiltration of the left lower lung".

Case No. 4

A twenty-six-year-old mulatto male referred to the Ear, Nose and Throat clinic of the San Juan City hospital by the Cardiologist with the tentative diagnosis of the vocal cord paralysis due to an enlarged heart. The patient has been hoarse all his life but more so during the past ten years. His cardiac diagnosis is: Inter-Auricular septal defect and cardiac enlargement. Laryngeal examination confirmed the suspect paralysis of the left vocal cord. The cord is paralyzed in the cadaveric position. It is flaccid and occupies a lower level than the normal right vocal cord. The voice is feeble. This case falls into the 10% of recurrent laryngeal nerve paralysis that in time undergoes complete paralysis due to atrophy of disuse of the cricothyroid muscle. The right vocal cord is normal. Chest plate reveals "Slight enlargement in the transverse diameter of the heart which in oblique projection is seen to be due to slight enlargement of the left ventricle associated with promi-

nence of the pulmonary conus and artery segment. No other chamber enlargement is detected. The pulmonary vascularization is not unusual."

SUMMARY

The etiologic factors of true vocal cord paralysis are listed after consideration of the nerve supply to the larynx. Four cases of left vocal cord paralysis due to intra-thoracic pathology are reported. In two of these cases the diagnosis of pulmonary disease was first suspected by the laryngologist after examination of the larynx. In the third case the chest condition was recognized by the Internist but there was no suspicion of cord paralysis. In the fourth case the cardiologist suspected vocal cord paralysis and this was confirmed by laryngoscopy.

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Ricardo F. Fernández, M.D., Oftalmólogo; nació el 31 de mayo de 1909 en Aguadilla, P. R.; padres José Fernández Lisboa y Julia García Montes; esposa Margarita Marchese; hijos Ricardo, José Luis y Héctor; graduado de médico en George Washington University el 10 de junio de 1931; internado en el Hospital Municipal de San Juan; residente en el Hospital Municipal de Arecibo, 1933-34; estudios postgraduados en oftalmología, New York Postgraduate Medical School, 1934; asistente de su hermano, el doctor Luis J. Fernández, hasta el 1938; primer oftalmólogo puertorriqueño en aprobar el Board americano de la especialidad 1938; cirujano residente del Instituto Oftálmico de Puerto Rico hasta el 1946; actualmente Director médico de dicha institución; miembro A.M.P.R., A.M.A., Acad. Oftal. Americana; Colegio Americano de Cirujanos, Colegio Internacional de Cirujanos; Asoc. Panamericana de Oftalmología; Vicepresidente, Asoc. Panamericana de Oftalmología; expresidente, Sección de Oftalmología de la A.M.P.R., Asoc. Médica del Distrito Norte.

Tal es, a grandes rasgos, el médico que ocupa hoy la presidencia de la Asociación Médica de Puerto Rico. Su interés en los problemas médicos del país y sus actuaciones en pro de la clase médica son bien conocidas de todos nuestros lectores. Resulta pues, innecesario que hagamos un recuento de ellas en esta breve nota. Preferible es que aprovechemos esta ocasión para recordar a nuestros compañeros cuál es el programa de nuestro presidente actual, según lo ha expuesto en su primera carta mensual, y que exhortemos a todos a darle el más decidido respaldo para que pueda triunfar en la árdua labor que le hemos impuesto. He aquí su programa:

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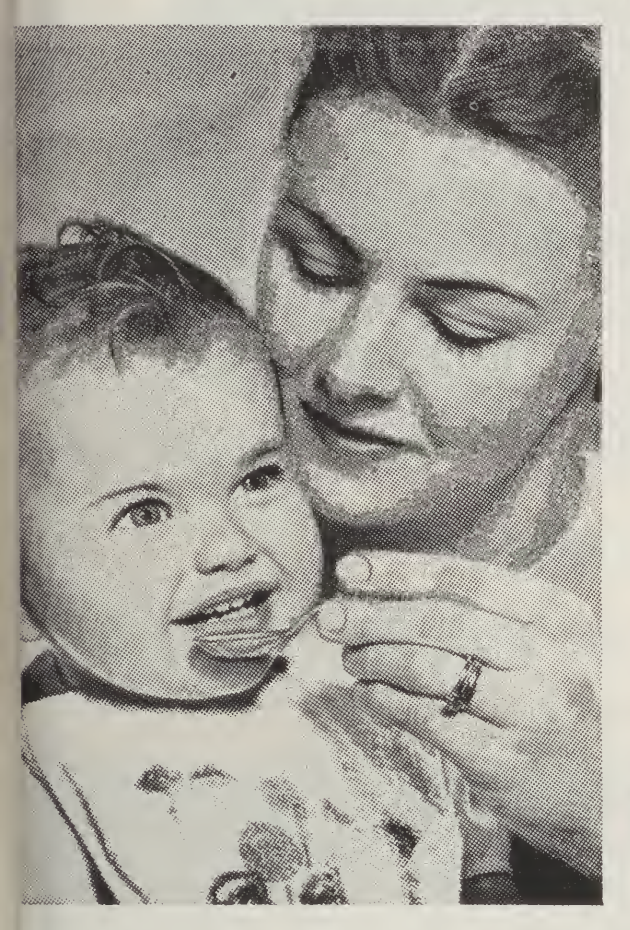
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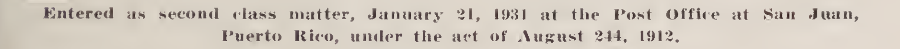
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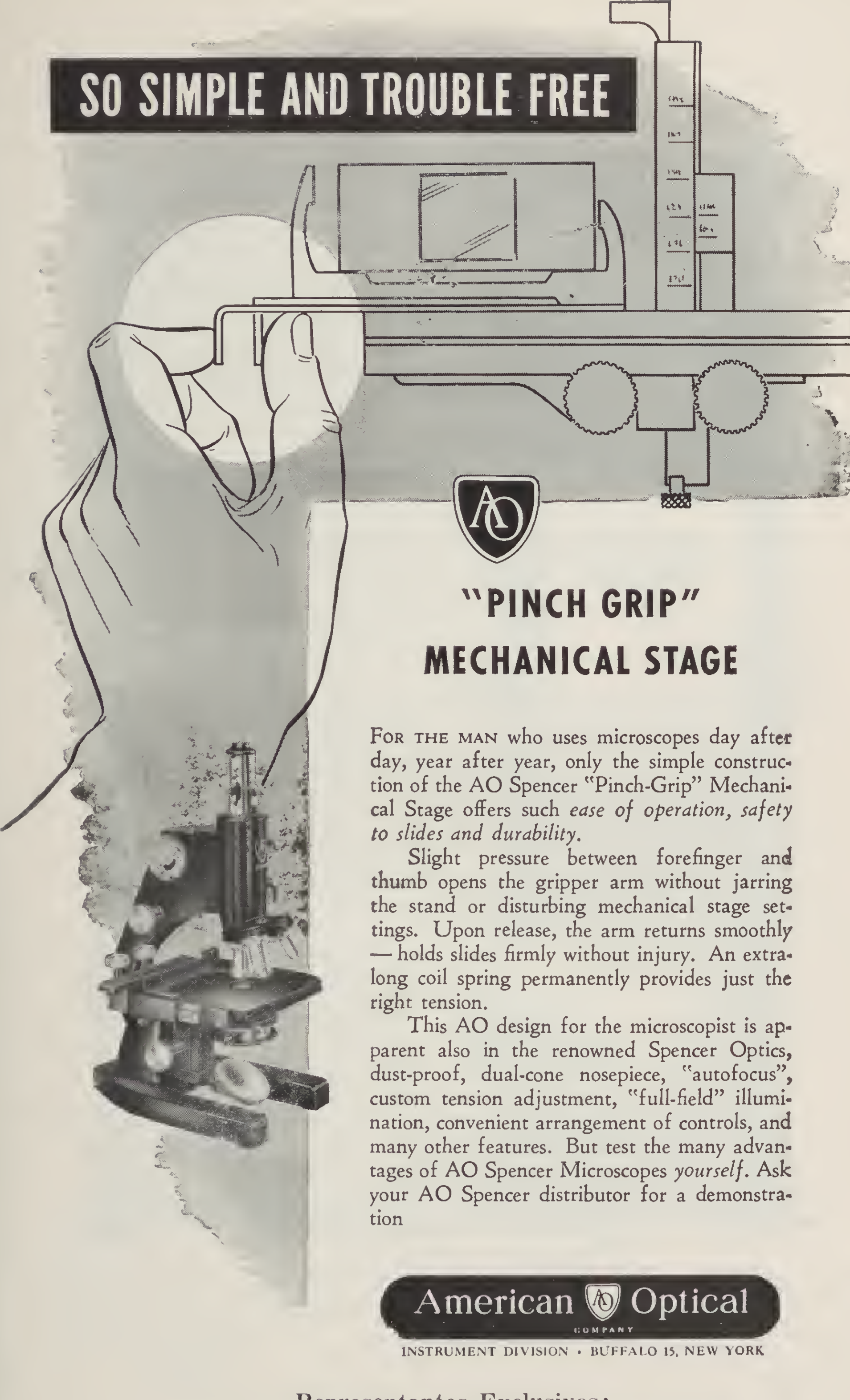
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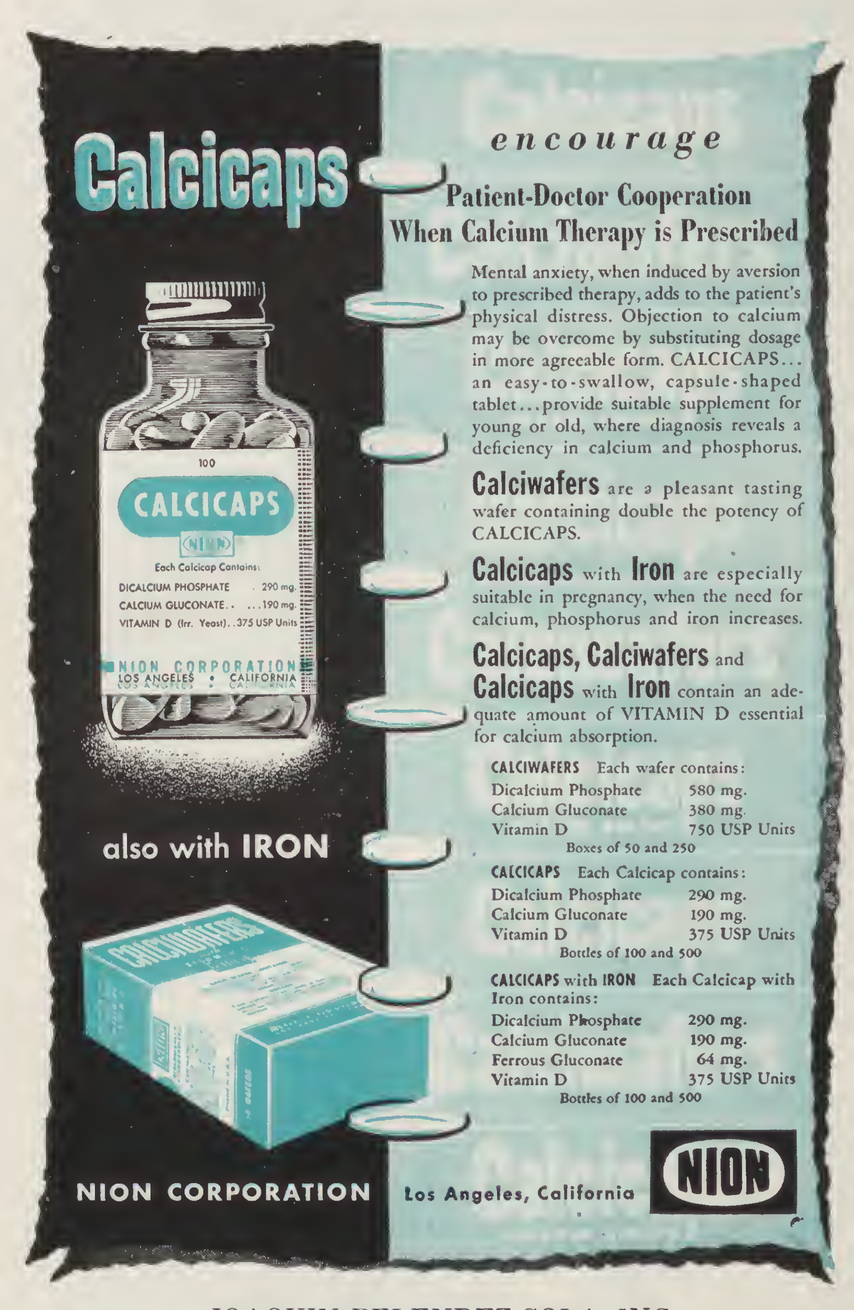
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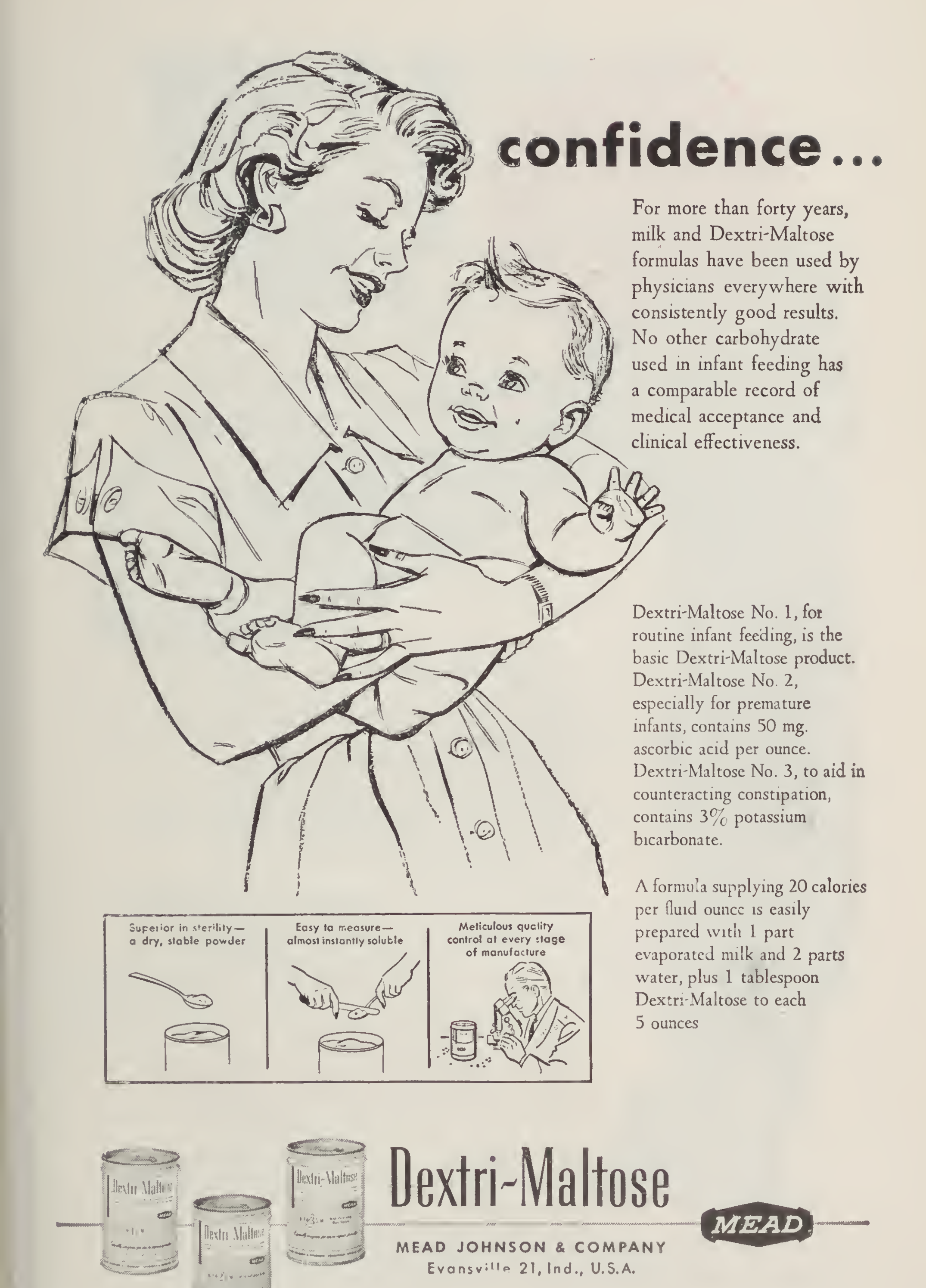
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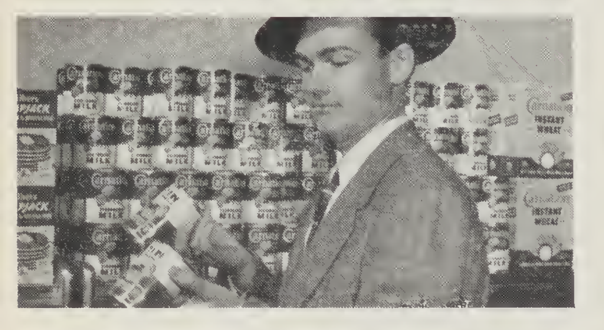
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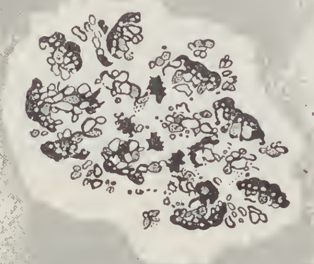




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Ampuls, 5 mg., 1 cc.: packages of 5.
Tablets, 1 mg. (s. c. orange): bottles of 50.
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• preoperatively to control oozing during surgery and provide clearer operative fi
one to two ampuls (5 to 10 mg.) every two hours prior to surgery for two doses.

• postoperatively to prevent hemorrhage and check oozing:
one to two ampuls (5 to 10 mg.) every two hours until there is no indication of un
bleeding. To maintain control, one ampul may be administered every three hours
1 to 5 mg. orally t.i.d.

• to control active bleeding:
one ampul (5 mg.) every two hours until bleeding is controlled; frequency of dose m
then be diminished.

• to control mild, low-grade bleeding:
one ampul (5 mg.) every three or four hours until bleeding is controlled; then 1 to 5
orally four to five times daily until bleeding ceases.

• maintenance dosage to prevent bleeding in conditions where small vessel integrity m
be impaired:
1 to 5 mg. orally t.i.d.; if bleeding ensues, dosage may be increased 1 to 5 mg. orally ev
three or four hours; if bleeding persists, oral dosage should be supplemented with
ampul (5 mg.) daily.

• pediatric dosage:
up to four years of age—1 mg. intramuscularly or orally with same frequency as
adults, until bleeding is controlled. From four to twelve years of age—one-half the
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THE PROBLEM OF RHEUMATIC ACTIVITY*

EVALUATION OF SOME LABORATORY METHODS

*RAMON M. SUÁREZ and RAMON M. SUÁREZ JR.***

(With the technical collaboration of Mr. Luis Saldaña)

The diagnostic criteria for rheumatic fever as given by Jones[1] in 1944 still hold good. These criteria divide the manifestations of rheumatic fever into major and minor manifestations.

Among the major manifestations, Jones includes carditis, arthralgia, chorea, subcutaneous nodules, and a verified history of previous rheumatic fever. For the latter, there are fever, abdominal or precordial pain, erthema marginatum, epistaxis, pulmonary changes, and laboratory abnormalities. As laboratory abnormalities, Jones mentions electrocardiographic changes, microcytic anemia, elevated leucocyte count, and an increased sedimentation rate. Diagnosis can be established by any combination of the major manifestations, or by the combination of at least one major and two minor manifestations. He also claims, and rightly so, that the presence of rheumatic heart disease increases the diagnostic significance of the minor manifestations, when no other cause for such manifestations exists.

With these diagnostic criteria in mind, it is relatively easy to establish a diagnosis of rheumatic fever. However, after the diagnosis has been established and the patient is under medical management, and/or has developed valvular heart disease and surgical intervention is being contemplated, the detection of activity becomes an important problem.

In some patients there will remain always obvious clinical signs and symptoms of active rheumatic fever, and these should settle the matter. For others, the laboratory has proved of some help. The fact that over a dozen laboratory tests are being presently used to determine rheumatic activity seems to indicate that there is still ample ground and need for more specific and sensitive procedures.

Among the most commonly used laboratory tests for rheu-

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* Studies performed under the auspices, and in the laboratories of Fundación de Investigaciones Clínicas (FIC), Santurce, Puerto Rico.

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matic activity, I shall mention the following twelve but shall limit the discussion to our experience with the first five:

1. Total leucocyte count.
2. Erythrocyte sedimentation rate.
3. Serum antistreptolysin 0 titer.
4. Serum C-reactive protein.
5. Skin sensitivity to Trafuril ointment.
6. Weltmann serum coagulation reaction.
7. Determination of serum mucoprotein.
8. Measurement of nonspecific hyaluronidase inhibitor in the serum.
9. Measurement of serum complement.
10. The bactericidal activity of the blood versus **B. subtilis.**
11. A serum precipitation reaction with a quaternary ammonium salt.
12. Diphenylamine color reaction with serum.

Leucocytosis. A leucocytosis of 10,000 to 16,000 per cubic millimeter, caused by a relative and absolute increase in polymorphonuclear leucocytes, is a common finding in active rheumatic fever. There is a shift to the left in the number of nonsegmented neutrophils by the Schilling count. Prior use of penicillin and other antibiotics, as well as salicylate therapy, may lower the leucocyte count, but it is a well known fact that rheumatic activity may persist even after the leucocyte count has decreased to a normal figure. Leucocytosis, although useful, is therefore not a sensitive and reliable index of rheumatic activity.

Erythrocyte sedimentation rate. The sedimentation rate of red blood cells increases in rheumatic fever, because of increased rouleaux formation of the erythrocytes. In one hour the column of plasma by the Westergren's method may rise from 90 to 130 mm. Values below 15 mm. are considered normal. Due to its simplicity and relative reliability, this test is the most widely used single test for rheumatic activity. It has proved useful as an index of the progress and adequacy of supressive therapy.

Its deficiencies and limitations are numerous, however. In the first place, an increase in sedimentation rate is not specific for rheumatic fever, for it is a common finding in most infectious diseases. It may be normal or equivocal in the presence of low grade activity, but becomes normal in the presence of successful salicylate or hormonal therapy. It may, in some cases, remain increased long after all other evidences of activity has disappeared, and fall to normal in congestive heart failure, even in the presence of active infection.

Antistreptolysin 0 titer. The first studies of antistreptolysin titer carried out in Puerto Rico were those of Morales Otero and

Pomales, at the School of Tropical Medicine. Their studies were mainly related to cases of recurrent tropical lymphangitis. The test is simple to accomplish now that the reagent, streptolysin 0 antigen, has become commercially available.[†]

We performed 218 such tests in 1953 (Fig. 1) and an additional 32 in 1954, or a total of 250. Our results in a group of 105 healthy adults, 52 healthy children, 33 cases of rheumatic valvular heart disease, and 28 cases of miscellaneous diseases, including one case of acute rheumatoid arthritis, were reported at the annual meeting of the Puerto Rico Medical Society in December 1953, but they have not yet been published.

There was no significant difference between the antistreptolysin 0 titers found in healthy Puerto Ricans and those reported for similar groups in continental United States. It seems now that streptococcal infections are probably as frequent here as they are in more northern climates.

In the group of 105 healthy adults, the average antistreptolysin 0 titer was 55 units; in 52 healthy children, 133 units; in the group of patients suffering from various diseases, it was 152 units, and in 33 subjects showing rheumatic valvular heart disease (mostly clinically inactive), the average was 169 units.

FIG. 1_ ANTISTREPTOLYSIN 0 TITERS IN 218 PUERTO RICANS STUDIED IN 1953.

† Difco Laboratories, Detroit, Mich.

Several investigators[2, 3, 4] have discovered that infants and very young children show a low incidence of antibody response following streptococcal infections. In the group studied by us, there were no infants and no children below the age of 5, but it might be interesting to note that the antistreptolysin 0 titers were definitely lower in healthy adults than in healthy children. Fig. 1 shows that 76% of the adults and only 35% of the children showed titers below 50. If we consider titers of 250 or above to be abnormal, it will be observed that only 1% of the healthy adults, 13% of the healthy children, 18% of patients suffering from various ailments, and 8% of cases of rheumatic valvular disease showed high or abnormal antistreptolysin titers. In the two groups of healthy subjects, no titer higher than 333 units was observed. In the group of miscellaneous diseases, one patient suffering from pneumonia showed a titer of 500 units, and a titer of 833 units was obtained in a man suffering from acute rheumatoid arthritis. The highest antistreptolysin 0 titer (1,250 units) was observed in a young man of 17 suffering from acute rheumatic carditis and valvular heart disease.

It has been established on sound clinical basis that a definite relationship exists between streptococcal infections and rheumatic fever. It has been shown within the last few months that a streptococcal proteinase isolated in 1950, produces severe damage to the heart muscle and valves without injuring or affecting other organs, when injected into the experimental animal. Wood and McCarty[5] reported that 92% of rheumatic fever patients studied gave antistreptolysin 0 titers of 250 units or above, a few going as high as 4,000 and more units.

Of 28 cases suspected of having rheumatic fever and referred to us for diagnosis and clinical evaluation, (Table 1), only six showed titers of 250 or above. C-reactive protein was present in five of the six cases.

Antistreptolysin 0 titers below 250 units were seen in 22 cases. C-reactive protein was present in 10 and absent in 12 of them. In none of the cases showing titers below 50 units could C-reactive protein be detected. There was, therefore, some correlation between the degree of antistreptolysin 0 titer and the presence or absence of C-reactive protein in the higher and in the lower group of antibody reactors, but there was a considerable number of patients in whom such correlation could not be established.

It is an accepted fact that the absence of a significant rise in the antibody titer does not exclude a recent streptococcal infection or rheumatic fever. The method has, therefore, many limitations. It is more useful in the diagnosis of early rheumatic fever, especially when an increase in the titer is obtained on re-

TABLE I

Cases	C-reactive Protein	Antistreptolysin 0 titers (Units)
1— R. F. G.	+	500
2— B. A. C.	++	250
3— R. J.	++	250
4— V. L.	++	250
5— R. F. G.	++	333
6— J. M. C.	0	250
7— N. L.	++	166
8— J. L. B.	+	125
9— J. M.	++	166
10— E. C. C.	0	100
11— R. R. O.	+	125
12— M. R.	+	125
13— I. H. G.	0	125
14— C. M. J.	++	100
15— E. G.	++	166
16— L. C.	+	100
17— K. O. A.	0	50
18— J. T. V.	+	50
19— F. C.	+	50
20— K. O.	0	50
21— E. S. A.	0	50
22— M. R.	0	12
23— I. J.	0	12
24— B. P.	0	12
25— C. L. A.	0	12
26— I. R.	0	12
27— M. N.	0	12
28— I. O.	0	12

peated testing of the patient's serum, but it is not practical for a definite determination of rheumatic activity.

C-reactive Protein. This protein, which is not normally present in the blood of human beings, appears in response to a number of inflammatory stimuli as well as in viral and neoplastic conditions, and may be identified by its capacity to form a precipitate with the somatic C-polysaccharide of the pneumococcus. Several investigators claim that it is at present the most sensitive test of rheumatic activity, and with the availability of the specific rabbit antiserum,† the test is being extensively used in various laboratories.

One of the advantages of the C-reactive protein test, according to Wood and McCarty,[5] is that "normal subjects give completely negative reactions and there is no 'normal range' to complicate interpretation."

It should be remembered that chorea, erythema marginatum,

† Produced commercially by Schieffelin and Co., New York.

subcutaneous nodules and Aschoff bodies in the auricular appendages may be present without causing the appearance of C-reactive protein and that adequate hormonal and salicylate therapy induces a return to normal of both the sedimentation rate and C-reactive protein.

Trafuril Skin Test. Influenced by the work of Nassim and Banner in England, Streitfeld and Saslaw,[8] of the National Children' Cardiac Hospital at Miami, Florida, studied a number of healthy children and children suffering from congenital and from active rheumatic disease. Skin response to the topical application of a vaseline-lanolin ointment, containing 5% of the rubefacient tetrahydrofurfuryl ester of nicotinic acid,[††] was tested.

A cutaneous hyperemia and/or edema was produced in normal subjects and in those with congenital heart disease or non-rheumatic conditions. Twenty of 22 patients with active rheumatic disease failed to show the typical hyperemia. Inactive rheumatics and patients whose activity had been supressed by cortisone or aminopyrine gave typical hyperemic reactions.

This test is so simple, innocuous, and rapid that, if confirmed as sensitive enough in a large enough number of cases it should prove of great practical help.

We have performed 25 such tests on the same number of patients suspected of rheumatic fever or of rheumatic heart disease. Only four failed to show skin hyperemia at the site of inunction and all four were found to be suffering from active rheumatic fever. The other showing definite skin hyperemia, spread zone, and/or edema had inactive valvular heart disease, or were suffering from diseases other than rheumatic fever.

Thirteen patients had leucocyte counts, sedimentation rate, determination of antistreptolysin 0 titer, and of C-reactive protein as well as skin sensitivity to **Trafuril** ointment. Table 2 shows the findings for the last three tests.

It will be seen that there is apparently no good correlation between the results obtained with the three tests. Cases 8 and 11 showing the highest antistreptolysin 0 titers (250 and 333 units, respectively) and the presence of C-reactive protein gave typical skin hyperemia, which is usually observed in normal or nonrheumatic subjects. Cases 1, 4, 6 and 7 showed absence of C- reactive protein, low antistreptolysin 0 titers indicative of inactive or suppressed rheumatic fever, but the skin test was typical in two and negative or atypical in the other two cases.

Case 1, a girl suffering from acute rheumatic carditis showing signs and symptoms of activity and who died shortly after,

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†† Supplied by Ciba Pharmaceutical Products, Summit, N. J., under the trade name of *Trafuril*.

TABLE II

Cases	C-reactive Protein	Antistreptolysin 0 titers (Units)	Trafuril Skin Reaction
1— M. R.	0	12	0
2— N. L.	++	166	+++
3— J. L. B.	+	125	++++
4— I. J.	0	12	++
5— J. M.	++	166	++
6— E. O.	0	50	++
7— E. C.	0	100	0
8— R. F.	++	333	++
9— R. R.	+	125	++
10— M. R.	+	125	+
11— V. L.	++	250	+++
12— B. P.	0	12	+++
13— C. A.	0	12	++

gave, while under adequate full or heavy salicylate therapy a negative C-reactive protein test, only 12 units of antistreptolysin titer, and no skin hyperemia with **Trafuril** ointment.

Conclusion. We have discussed the problem of rheumatic activity from the clinical and laboratory standpoints.

Although the leucocytic count, the sedimentation rate, the determination of streptococcal antibodies and C-reactive protein in the patient's serum, and the cutaneous test with **Trafuril** ointment may be of some aid there is undoubtedly need for more specific and sensitive tests to determine rheumatic activity.

A careful and thorough clinical study and evaluation remain the most important elements in the physician's final appraisal of rheumatic activity.

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THE TREATMENT OF PRIMARY TUBERCULOSIS IN CHILDREN

RESULTS OF A CONTROLLED STUDY USING ISONIAZID

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The advent of isoniazid as a new powerful drug against tuberculosis has brought new problems in regard to the indications for its use. Although it is well known that therapy of primary tuberculosis with streptomycin does not affect the course of a simple primary tuberculosis[1] nor prevent its most important complication, meningitis, the role of isoniazid in this regard is not clearly understood.

A brief review of the fate of the primary complex[2,3,4] may give an idea of what complications we would be trying to prevent by giving isoniazid to children with primary tuberculosis.

The primary complex may persist for several months and disappear usually after a year, leaving as a trace, calcifications in the site of the primary focus and in the draining lymph nodes. Commonly, during the second month after the appearance of the primary complex an occult hematogenous dissemination takes place. This may give rise to a moderate temperature elevation, lymphadenopathy and splenomegaly of a few days duration. This is responsible for many of the early complications of primary tuberculosis usually observed during the first year of the disease. Such complications include various manifestations of hematogenous tuberculosis such as tuberculous meningitis, renal and osseous tuberculosis. Hematogenous dissemination may be massive, thus producing miliary tuberculosis. It may be intermittent and of varying dosage thus producing the picture of prolonged intermittent unexplained fever which characterizes protracted hematogenous tuberculosis.

The primary lesion may progress locally by excavation and by bronchogenic dissemination. This form of locally progressive tuberculosis is called progressive primary tuberculosis. This should not be confused with the tracheobronchial compression produced by the lymph node enlargement associated with the primary complex. Such compression of the bronchi and trachea gives rise to endobronchial tuberculosis and symptoms such as severe cough,

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From the Department of Pediatrics of Alejandro Ruiz Soler Sanatorium* and the School of Medicine of the University of Puerto Rico.† This work was aided by a grant from the Committee on Medical Research of the American Trudeau Society, medical section of the National Tuberculosis Association.

wheezing, stridor and respiratory distress. This may be accompanied by shifting areas of atelectasis often labeled epituberculosis. These symptoms and signs are the result of mechanical factors and need not be associated with actual progression of the disease. For this reason the so called tracheobronchial form of primary tuberculosis shows a surprising lack of response to chemotherapy.[5]

Tuberculosis of the tracheobronchial lymph nodes may extend to other lymph nodes of the body via the lymphatics or by hematogenous dissemination. Common sites of invasion are the tonsils and the cervical lymph nodes. The middle ear is often invaded by direct extension through the eustachian tube from tuberculous adenoids. Thus it is not uncommon to observe children with cervical scrofuloderma, large tonsils and chronic otitis media.

Reinfection type of tuberculosis is not uncommon among our children. It may originate from endogenous or from exogenous sources. The endogenous type is the most common and occurs as a result of activation of a dormant focus in the pulmonary apices. This dormant focus is thought to have resulted from seeding to the apices at the time of occult hematogenous dissemination. In certain cases this apical focus may be observed in the X-Ray as a small apical calcification which has been labeled "Simon Focus".

The only way to determine whether isoniazid is effective in preventing the complications of primary tuberculosis would be to set up a carefully controlled study in which treated as well as untreated cases were observed under a similar environment.

The importance of such a study specially in Puerto Rico may be realized from an analysis of the number of children with primary tuberculosis encountered in our island. The population of Puerto Rico in the age group less than five years of age is around 336,000. Rodríguez Pastor and Janer[6] have reported 19% positive tuberculin reactions in this age group; Therefore, a discovery that isoniazid prevents the complications of primary tuberculosis would mean treating over 60,000 children in Puerto Rico before they reach five years of age.

Because of the tremendous proportions of such an undertaking a careful appraisal of its advantages and disadvantages is essential. The pros and cons may be summarized as follows: I—Arguments in favor of treating all children with primary TB. a)—Prevention of complications of primary TB. mainly tuberculous meningitis. b)—Possible prevention of endogenous re-infection TB. at a later date. II—Arguments against treatment of primary TB.

a)—It is not certain that such therapy prevents meningitis. Definitely, a short course for 3 mos. or less does not prevent meningitis. We have observed tuberculous meningitis in one patient who received a short course of streptomycin and isoniazid and we know of three other cases elsewhere.[7] b)—If a complication occurred in a previously treated patient the bacillus might have developed resistance to isoniazid and the patient would be unable to benefit from the drug.

MATERIALS AND METHOD

Children admitted to the Pediatrics Department of the A. Ruiz Soler Sanatorium were considered for the study. The following criteria were used for the selection of cases. 1.—Positive tuberculin. 2—Visible lesion on X-Ray of the chest either parenchymal or hilar without calcifications. 3—No evidence of cavitation or spread. In other words, children with recent, active, primary tuberculosis who did not have any other form of tuberculosis and no evidence of progressive primary tuberculosis were selected.

Selected cases were randomized by using tables of random numbers of Kendall and Smith; Cambridge University Press 1939. A control group was untreated. The other group was treated. The equivalent of the adult dose of 12 gms. of PAS and 300 mg. of INH was used. This dose was scaled down to children in terms of square meters of body surface. This is equal to 7.25 gms. of PAS and 180 mg. of INH/Sq. meter of body surface. Daily dosage was divided into three portions. One group of patients received 4 mos. and another group 12 mos. of therapy. The following laboratory procedures were performed in most of the cases. 1—X-Rays: P.A. and lateral films were made prior to therapy and P.A. films monthly during therapy. 2—Sputum or gastric concentrate with cultures were performed before therapy and at monthly intervals thereafter. 3—Urine: Urinalysis was performed within 2 weeks prior to therapy and monthly thereafter. 4—Renal function: NPN was done within two weeks prior to therapy and during the fourth month and 12th. month of therapy. 5—Blood: CBC was performed within 2 weeks prior to therapy and at monthly intervals during therapy. 6—Weight: The patient was weighed within 2 weeks prior to therapy and at monthly intervals during therapy.

Patients were followed up in the hospital for a one year period and an attempt was made to continue this follow up for a longer period of time (Table I). Tuberculin test was performed at the completion of therapy and at the end of one year follow up.

TABLE I

DURATION OF OBSERVATION

MONTHS:	2-3	3-6	6-9	9-12	12-15	15-21
UNTREATED TOTAL 23	2	6	2	3	—	10
TREATED 4 MO. TOTAL 14				3	5	6
TREATED 1 YR. TOTAL 15	3	8	3	1		

RESULTS

Untreated Group (23 patients)

All patients in this group are alive; more than half of these patients have been observed for 1 year or more. All the patients except one, had persistent pulmonary lesions with gradual regressive changes.

Three were removed from the study because of the following complications which necessitated treatment with isoniazid:

(1) progressive primary tuberculosis (2) extensive endobronchial lesions with severe toxicity (3) tuberculosis of cervical lymph nodes with abscess and sinus formation. No patient has developed tuberculous meningitis yet. All complications occurred during the first year of observation.

Treated 4 mos. (14 patients)

All these patients are alive. The pulmonary lesions persisted during therapy in all, but cleared on the fourth and fifth trimesters of observation in four patients. One patient was restarted on isoniazid on the seventh month of observation because of severe endobronchial lesions with toxicity. In other words: four months of chemotherapy did not prevent endobronchial lesions.

Treated 1 yr. (15 patients)

All these patients are still under observation except one who died on the third month of therapy from purulent meningitis (definitely not tuberculous on autopsy); however, the pulmonary, lesion was progressive primary with presence of mycobacterium tuberculosis on culture of lung material. Another patient in this group developed endobronchial lesions during the first trimester

of chemotherapy which persisted for 6 mos. despite the drugs. The pulmonary lesions are clearing but persist in all but two of the patients.

The temperature and weight of all groups was similar except for those patients who developed complications in whom temperature elevation and failure to gain weight were noted.

No conversions of the tuberculin reaction to negative have been observed yet.

Blood studies, hepatic and renal function tests revealed no abnormalities related to the drug therapy.

DISCUSSION

We have no final answer for the problem of the desirability of chemotherapy of uncomplicated primary tuberculosis.

The main complication which we are trying to prevent, meningitis, has not been observed in either treated or untreated groups.

It has been estimated by Miller[8] that the incidence of tuberculous meningitis among patients suffering from active primary tuberculosis is around 3%. In order to prove that a given drug is 100% effective in preventing meningitis and other complications of primary tuberculosis it would be necessary to observe around 700 cases approximately, half of whom would serve as controls and the other would be treated.

In our comparatively small group we have observed that complications occurred in three of untreated patients and in one of the patients following a short course of chemotherapy. No patient in the one year of chemotherapy group has had any complications, though it was shown, in the one who died on the third month of therapy from a nontuberculous illness, that a progressive primary lesion was present at this early stage of therapy. The roentgenographic changes in both treated and untreated groups are similar although there is a suggestion that the treated patients may have shown clearing at a slightly faster rate. However, chemotherapy did not prevent endobronchial lesions.

The evidence we have so far is not statistically significant and therefore insufficient to allow us to recommend chemotherapy for all patients with active primary tuberculosis or recent tuberculin conversion. However, it does serve to demonstrate the importance of such a study and the desirability of observing a sufficiently large number of patients to provide a statistically significant answer.

For this reason we are about to start a study sponsored by the National Tuberculosis Association and the United States Public

Health Service[9] in cooperation with twenty centers in the United States so that the final answer may be available perhaps within the next two years instead of the five or ten years it would take in an isolated center.

CONCLUSIONS

The question as to whether all children with uncomplicated active primary tuberculosis should receive isoniazid is not settled yet.

In our series of 29 treated patients and 23 controls, the treated patients had fewer complications and slightly faster roentgenographic clearing. Tuberculous meningitis did not develop in either group. The small number of cases in this study provides results that are not statistically significant; therefore, the investigation should be continued on a much large scale.

RESUMEN Y CONCLUSIONES

Se estudió el tratamiento de la tuberculosis primaria activa con isoniacida y PAS. Se trataron 29 enfermos mientras que otros 23 fueron casos testigos sin quimioterapia. No se observó meningitis tuberculosa en ninguno de los dos grupos, pero los enfermos que recibieron isoniacida y PAS tuvieron menos complicaciones.

A pesar de estas observaciones no es posible todavía, llegar a conclusiones finales sin un aporte casuístico de mayor volumen.

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INDUCCIÓN DEL PARTO

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Entendemos por inducción del parto la terminación artificial de un embarazo por métodos médicos, quirúrgicos o mecánicos. La primera inducción de parto registrada en tiempos modernos data del año 1738 en Inglaterra cuando una comadrona rompió prematuramente las membranas a una parturienta con estrechez pélvica. Como el mecanismo exacto por el cual se inician los dolores del parto es aún desconocido los métodos para inducir el trabajo del parto son variados y empíricos. Desde ese tiempo hasta que se generalizó la operación cesárea como procedimiento seguro, la principal indicación para la inducción del parto fué la desproporción cefalopélvica. A principios del siglo 19 Kluge empezó a inducir partos introduciendo gasas dentro del cuello uterino; Simpson y Scanzoni inyectaban ácido carbónico en el útero; Krause en 1855 preconizó el uso de las bujías de goma introducidas entre las membranas y la pared uterina; y en el 1888 aparecieron los balones de Champetier de Ribes más tarde modificados por Voorhees. En 1925 se empezó a usar una vejiga de oveja que se llenaba de glicerina después de introducida en el cuello uterino. En cuanto a los procedimientos médicos se usó por muchos años la administración de aceite de castor, enemas y una sal soluble de quinina el cual fué abandonado por haberse producido complicaciones como la sordera fetal y muertes fetales cuya causa fué sin duda la administración de este medicamento. Desde principios de siglo todos estos procedimientos fueron prácticamente abandonados habiendo imperado desde entonces como medio más seguro y eficaz entre los procedimientos mecánicos la rotura artificial de las membranas. En 1909 Bell por vez primera preconizó el uso de la pituitrina para la inducción del parto por vía parenteral. En 1912 Hofbauer recomendaba el uso de la pituitrina intranasal y posteriormente el uso del principio ocitócico por vía intravenosa.

En la actualidad los métodos comunmente usados para inducir el trabajo del parto se han reducido a tres:

1 —Rotura artificial de las membranas.
2— Administración de pitocina.
3— Combinación de ambos.

Cuando un médico se decide a inducir un trabajo de parto debe tener presente ante todo que es responsable por dos vidas; la madre y el niño por nacer, y debe recordar que toda parturienta debe abandonar la sala de partos habiendo sufrido el menor trau-

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matismo posible. Debe también tener presente según Gibson que la inducción del trabajo del parto es una operación obstétrica mayor y que debe dársele la misma importancia y deben tomarse las mismas precauciones que cuando el cirujano decide hacer una operación de cirugía mayor. Hay sin duda un sinnúmero de indicaciones para la terminación de un embarazo. Dichas indicaciones pueden ser maternas o fetales o una combinación de ambas. La decisión para terminar artificialmente un embarazo es siempre difícil de hacer y depende en gran parte del criterio obstétrico adquirido por la experiencia y observación de muchos años. Debe tenerse siempre presente las razones, los motivos y la condición individual de cada caso.

Las principales indicaciones son las siguientes:

1—Rotura espontánea de las membranas sin que la mujer inicie el trabajo del parto en las próximas horas estando el cuello maduro. Debe tratarse de terminar dichos partos para proteger la parturienta de la falta de defensas contra una infección como supone un saco roto por mucho tiempo. Si el cuello no tiene la preparación necesaria es preferible esperar, ya que generalmente el trabajo del parto se inicia en un tiempo razonable y corto.

2—Separación precoz y prematura de la placenta. En estos casos si la hemorragia es masiva indicaría la terminación del embarazo por cesárea. La inducción del trabajo del parto si la hemorragia es moderada generalmente disminuye la pérdida sanguínea.

3—En pre-eclampsia está indicado cuando esta condición ha sido tratada médicamente sin que la paciente haya respondido al tratamiento administrado, manteniéndose los síntomas de toxemia.

4—Hidramnios o preñez múltiple, sobre todo cuando estos casos producen trastornos respiratorios o cardíacos.

5—Enfermedades orgánicas como la tuberculosis, diabetes, las cardiopatías y las pielitis recurrentes.

6—Placenta previa marginal. En estos casos debe siempre reemplazarse la sangre perdida.

7—Eritroblastosis fetal.

8—Muerte habitual del feto.

9—Anormalidades fetales incompatibles con la vida tales como la muerte del feto, peso excesivo del feto, hidrocefalia y anencefalia.

10—Hipermadurez fetal.

11—Conveniencia del médico y la paciente. Esta condición constituye la inducción electiva tan de moda en algunos hospitales, aceptada por muchos médicos últimamente, creando lo que se ha conocido como los niños por cita. Es una indicación sumamente discutible ya que no debemos anteponer la conveniencia del médico

o la paciente misma a la seguridad de la madre y el niño. El único factor que debe considerarse en esta indicación es la historia de partos rápidos cuando las distancias hasta el hospital son considerables en cuyo caso sería ideal que la madre entrara al hospital en un día determinado y pudiera empezar su trabajo de parto en condiciones también ideales.

Una vez determinada y establecida la indicación para la inducción del parto ¿qué requisitos debe reunir el caso para obtener resultados satisfactorios? En primer lugar hay que determinar la condición del cuello uterino. El cuello uterino debe estar maduro lo que implica que debe estar parcialmente dilatado y borrado. Un cuello largo firme y cerrado es el factor principal en los fracasos de la inducción. Puede estimularse la contracción uterina pero el cuello no se retrae y si pasadas varias horas de buenas contracciones no se observa alguna dilatación el proceso se considera un fracaso. No debe haber desproporción cefalo-pélvica. El feto debe estar muy cerca del término y debe estar de vértice. La presentación debe estar en la pelvis y por lo menos parcialmente encajada. Nunca debe intentarse una inducción en una presentación de cara, de frente o transversa. Intentarla rompiendo las membranas cuando la presentación aún está flotando siempre trae consigo el peligro de que se produzca un prolapso del cordón. No debe haber condiciones patológicas pélvicas; los quistes ováricos pueden interferir con la entrada de la presentación en la pelvis o romperse con el trauma del parto dando lugar a hemorragia o infección. Los tumores sólidos del ovario y los miomas pueden igualmente intervenir con el desarrollo normal del feto. Las pacientes que han sufrido traquelorrafias o perineorrafias tampoco son buenas candidatas para inducción. Si hay algún inconveniente para el parto natural es preferible considerar la posibilidad de una cesárea.

La paciente debe estar hospitalizada antes de iniciarse el procedimiento escogido y el médico debe estar preparado y equipado para cualquier clase de emergencia que surja. El caso debe ser admitido temprano por la mañana, preferiblemente en ayunas o la tarde anterior administrándosele algún sedante durante la noche. Debe ser preparada debidamente para un examen vaginal estéril y administrársele una enema de limpieza. Algunos recomiendan vitamina K. Si las condiciones que encontramos en dicho examen son los requisitos que enumeramos anteriormente procedemos a usar el método seleccionado. Si se trata de romper las membranas basta con introducir una pinza curva y hacer una tracción moderada de la bolsa hasta que fluya el líquido amniótico. Si las contracciones no se inician dos horas después debe iniciarse la administración de pitocina. Cuando se usa la pitocina por vía parenteral debe usarse en dosis muy limitadas, preferiblemente dos

mínimas cada 20 a 30 minutos por cinco dosis consecutivas. Cuando se usa por vía intravenosa el método más corriente es la administración de un centímetro cúbico de pitocina en 1000 cms. cúbicos de suero glucosado al 5% inyectado a razón de 10 a 15 gotas por minuto en su inicio y más tarde regulándose según la intensidad y la frecuencia de los dolores cuando éstos se presenten. Una vez hecho el procedimiento que hayamos seleccionado generalmente transcurre un período variable hasta que se inician los dolores; este período se conoce con el nombre de período latente. Una vez iniciado el trabajo de dilatación el parto generalmente transcurre sin inconveniente alguno. En los estudios hechos por Burnett en Newark, Cosgrove en Jersey City, Pomerance en Brooklyn, Kemwick en Pittsburgh, Ferguson en New Orleans, Fulsher en Hospital San Vicente en Portland; Hellman, Harris y Reynolds en Johns Hopkins; etc., los resultados han sido muy similares y todos concuerdan en que el tiempo total del parto ha sido más corto que en los casos de partos normales. Todos citan como complicación más frecuente el prolapso del cordón el cual ocurre a veces cuando se rompen las membranas sin estar la presentación en la pelvis.

Revisamos los casos de obstetricia del Hospital Susoni en el último año. Hemos procedido a practicar solamente siete inducciones.

En dos casos la indicación fué la hipermadurez fetal. En ambos se trataba del segundo embarazo, habiendo tenido el primer niño trastornos convulsivos serios en un caso y nacido muerto en el otro. Se le administró un purgante de castor y tan pronto fué efectivo se administró pitocina I. V. Las contracciones se iniciaron a las dos horas en un caso y a las 4 horas en el otro terminando el trabajo de parto en siete horas en ambos.

El tercer caso se trataba de un hidramnios agudo con monstruosidad anencefálica en una primeriza de 7 meses y medio de embarazo. Se hizo una rotura artificial de las membranas que alivió a la paciente de una gran distensión abdominal. Los dolores se iniciaron a las 4 horas y el parto duró 15 horas en total.

El cuarto caso era una multípara con embarazo de 8 meses cuyas membranas se habían roto espontáneamente en su casa el día anterior a su admisión. Se usó pituitrina intranasal. Las contracciones se iniciaron antes de la hora y el parto duró 3 horas y media.

El 5to. caso era una primeriza con hipermadurez fetal y monstruosidad anencefálica demostrada en estudios radiográficos. En esta paciente iniciamos el tratamiento sin que el cuello reuniera las condiciones necesarias ya que estaba completamente cerrado y del largo normal pero nos indujo a hacerlo el estado de ansiedad en que se encontraba la familia una vez enterados de las con-

diciones del feto. Usamos pitocina intravenosa y el primer día no obtuvimos más que unas contracciones leves que hicieron borrar parcialmente el cuello. Sedamos la paciente y al día siguiente administramos la segunda infusión después de una enema efectiva a las 9 de la mañana. Las contracciones se iniciaron a las 7 de la tarde y hubo que terminar el parto con una versión podálica a la una de la madrugada prolongándose por tanto solamente 6 horas.

El sexto caso era una multípara con pre-eclampsia que recibió tratamiento médico intensivo por dos semanas sin que consiguiéraramos disminuir la toxemia. Se hizo rotura de las membranas iniciándose las contracciones a las 4 horas y terminó su parto en dos horas y 10 minutos.

El 7º y último caso de esta corta serie se refiere a una multípara sin partos en los últimos 13 años. Fué admitida al hospital con falsos dolores de parto a los 13 días de haberse cumplido el término. Los alegados dolores desaparecieron pero la paciente permaneció hospitalizada por presentar edema de las extremidades y albuminuria que oscilaba entre 2 y 3 cruces. Su presión arterial era normal. El cuarto día estaba intranquila y ansiosa por cuya razón se decidió proceder a inducir el parto. Su cuello estaba blando y parcialmente borrado. Se usó pitocina intravenosa según la técnica ya descrita. Las contracciones se iniciaron a la hora y el parto terminó 8 horas más tarde.

El único natimuerto en los siete casos fué el anencéfalo que se hizo complicar de hidramnios. Todos los demás nacieron en buenas condiciones incluso la anencéfala hipermadura que vivió por 3 días.

Aunque los resultados de esta corta serie no nos autorizan a tirar conclusiones no hay duda que los mismos son idénticos a los observados en otros hospitales de mayor movimiento y podemos concluir que disponemos con la inducción del trabajo del parto de un método eficaz y satisfactorio para resolver muchas condiciones obstétricas. Dicho método debe usarse siempre estando la paciente hospitalizada; deben observarse cuidadosamente los requisitos necesarios para poner en práctica, debemos determinar juiciosamente la indicación olvidándonos de nuestra comodidad o de las conveniencias de la paciente; y los métodos de elección deben ser o la rotura artificial de las membranas o la administración intravenosa de pitocina.

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OLIGURIA AND ANURIA IN OBSTETRICS

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I have been given the privilege of addressing you on a topic which will affect every physician, and particularly the obstetrician, sometime during his professional activities, namely, the subject of oliguria and anuria particularly in reference to obstetrics. Ever since the clinical and pathological features of lower nephron nephrosis were described by Lucke, during the war, there has been renewed interest in this condition. It has been generally conceded in the past that hemorrhagic accidents frequently complicate toxemia of pregnancy and contribute to the production of renal shutdown that ultimately leads to renal insufficiency. Similarly, acute renal failure has been recognized for some years in obstetrical cases suffering from eclampsia, prolonged labor, sepsis and hemorrhage, mismanagement and abortion. The similarity of the clinical course in these patients to that found in trauma with shock, war wounds, burns and exogenous poisons has been noted by many writers. The treatment of the condition has varied over the years and at present is generally agreed upon.

There are a variety of predisposing factors for the production of renal complications in the obstetrical patient. These include 1. abruptio placentae, 2. placenta previa, 3. eclampsia, 4. sepsis of labor and abortion, 5. shock and trauma during labor, 6. post operative shock and post partum hemorrhage, 7. incompatible blood transfusion. The mechanism of the production of renal shutdown in these patients has been the subject of intense investigation in the U. S. and abroad, and several theories have naturally arisen from them. One of them, exposed by Sophian, indicts concealed intrauterine hemorrhage as the initiating factor. He postulates that compression of the pregnant uterus causes a reflex spasm of the efferent glomerular arterioles. A renal pressor substance is formed which produces cortical ischemia and tubular damage due to anoxia.

The constriction of the efferent glomerular arterioles effectively prevents the circulation in the glomeruli and the majority of the circulation is shunted thru the juxtamedullary lake, apparently producing afferent arteriolar spasm. This results in a fall in filtration pressure thereby preventing the nephron from performing its function of selective absorption and secretion and anuria supervenes.

A recent communication by McKay, Weiner, Hertig and Reid, from Boston, discussing the pathologic anatomy of eclampsia,

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bilateral cortical necrosis, and other acute fatal complications of pregnancy, in pointing out the similarity of the pathological lesions in these conditions to those of the Schwartzman phenomenon, suggests the theory of intravascular fibrin deposition in arterioles and capillaries throughout the body as the basic pathologic feature of the condition. They have extensively studied the pathologic anatomy of eclampsia, bilateral cortical necrosis of the kidneys, and pituitary necrosis in post partal women. From the pathological angle, several points of interest were demonstrated: Periportal fibrin deposition with hemorrhage and necrosis, characteristic of eclampsia, were found in patients with convulsions, as well as in anuric patients with bilateral renal cortical necrosis in which there were fibrin thrombi in the glomerular capillaries. These patients had similar thromboses in other organs of the body most commonly the liver, spleen, kidneys, adrenals & pituitary, with resultant hemorrhage and necrosis. They also noted that these fibrinous thrombi in small vessels could be found in the great majority of patients who died of acute complications of toxemias. In addition they described the pathologic changes in the placenta and decidua as consisting of placental infarcts, masses of laminated clot, and, as the most consistent change, the excess numbers of clumped syncytial nuclei (syncytial knots) suggesting, in general, a premature senescence of the trophoblast. Again, the vascular changes are those of fibrinoid degeneration of the walls of the spiral arterioles, with resultant thrombosis causing necrosis of the decidua and contributing to the occurrence of premature separation of the placenta. To add further weight to their theory, which I will summarize presently, certain hematologic observations in patients with abruptio placentae support the idea of intravascular fibrin deposition as a major event in these conditions. A clotting defect in these patients with abrubtio placentae, caused by a rapid drop in circulating fibrinogen has been designated afibrinogenemia, and its basic cause is due to a sudden loss of fibrinogen due to the intravascular deposition of this substance in the form of fibrin. It would appear that a toxic substance from the uterine contents, which has the ability to reduce fibrinogen is released into the circulation in cases of abruptio placentae. The pathological changes of fibrin thrombi described above have been observed in animals being subjected to the Schwartzman phenomenon, and indeed, in these animals the lesions observed are in effect, bilateral cortical necrosis of the kidneys, with anuria, which is exactly the same lesion found in the toxemia of pregnancy. In summary, they offer their explanation as follows: toxic material suddenly injected into the maternal blood stream, and arising from the intrauterine contents, is responsible for the deposition of fibrin,

the hemorrhages and necroses in the renal cortices, the liver, spleen, adrenals and other organs, and is responsible for the clinical symptoms of shock and anuria in patients suffering from eclampsia, abruptio placentae, sepsis of labor, prolonged labor, etc., and that these pathologic changes are manifestations of the generalized Schwartzman phenomenon in human beings. In some cases the toxin may be a bacterial toxin while in others, particularly those associated with toxemia, the toxin may be similar to the now partially discredited "menstrual toxin" of Smith and Smith.

So much for the theories as to the causation of the kidney lesions of bilateral renal cortical necrosis, or necrotizing papillitis, and lower nephron nephrosis, which are the main pathological entities, giving the picture of oliguria and anuria in obstetrical patients.

The clinical picture in these cases is usually dramatic and not infrequently tragic, depending on the extent of the renal pathology. The onset of oliguria and/or anuria closely follows the precipitating episode; and lasts variously from two to twenty or more days. If recovery occurs it usually does so in an average of six to seven days, as evidenced by a gratifying diuresis. The time of onset of diuresis is usually directly proportional to the extent of the renal lesion. Diuresis ensuing prior to the 10th day usually forecasts a favorable prognosis. There is usually an output of 30-60 cc of grossly bloody urine, or none at all. The presence of gross blood is not necessarily pathognomic of cortical necrosis, as stated by some writers. The specific gravity of the urine is low and fixed. Cellular and granular casts frequently appear in the urine. The presence of heme casts is a frequent finding if properly looked for.

In all cases in which renal failure persists for more than 2 days there is azotemia and an increase in the blood urea nitrogen, NPN and other nitrogenous wastes. Alteration in carbon dioxide combining power, chlorides, potassium and sodium are frequent but may be controlled by therapy. Repeated flame photometer estimations of serum sodium and potassium are essential in management, as well as daily electrocardiograms for signs of potassium unbalance, which usually manifests itself in hyperkalemia.

In the treatment of anuria and similar states, the precipitating factor or factors must be treated first. The necessity of preventing shock and trauma due to prolonged labor, mismanagement and sepsis is imperative. The prompt replacement of blood in all obstetrical hemorrhage needs emphasis, since its importance is still unrealized by many obstetricians. This is particularly true in cases of abruptio placentae. Priddle, Stevenson, who have reported a series of these cases, contend that prompt blood replace-

ment following the occurrence of shock will lower the incidence of renal insufficiency. Even if the oliguric or anuric state has been already established, and shock persists, it should be still treated with transfusions of whole blood properly and carefully cross-matched.

Once the syndrome has been established, it is best managed conservatively, as emphasized by several investigators. Several reports in the literature, as well as clinical evidence to this effect indicate that death in lower nephron nephrosis and allied conditions, after the initial phase and before the onset of diuresis, is almost invariably the result of drowning the patient with excessive fluids. It appears that the caloric intake during the stage of oliguria and anuria is of secondary importance. Once we have a patient with an established syndrome, the only treatment that will offer any chance of recovery is conservative replacement of fluids and proper maintenance of electrolyte balance. Strauss, in his excellent monograph, stresses the danger of overloading the patient with fluids thereby producing pulmonary edema and death. He recommends the replacement of actual fluid loss only, and a diet of 100 Gm. glucose daily, because of its protein-sparing action, until the stage of diuresis is reached, when fluids and electrolytes are replaced volume for volume as reflected in the urinary output. Hyperkalemia can occur quite rapidly during the anuric stage and cause death and should be carefully watched for. It is the consensus of opinion that the human organism can be kept alive for 4 or more weeks in the presence of non-functioning kidneys if this regime is followed. It should be remembered that more caution in parenteral therapy is required for the toxemic edematous patient, since edema reduces the available storage space for excessive fluids and increases the possibility of development of pulmonary edema. When this overloading results, phlebotomy may be a life saving measure.

Diuresis usually begins between the second and twelfth day of oliguria. Fluids should then be replaced volume by volume and electrolytes maintained in balance . The electrolyte unbalance in this stage is manifested by salt depletion, and sodium chloride should be given in sufficient quantities to replenish the loses.

We feel, and this seems to be the present consensus of opinion, that radical measures to initiate diuresis are unphysiological and may be detrimental to the final outcome. We refer to such measures as capsulotomy (decapsulation of kidneys), peritoneal dyalisis, transplantation of kidneys, sympathetic block and caudal anesthesia, intestinal dyalisis, and artificial kidneys. All methods of dyalisis require great attention and a degree of surgical skill which may not be available at all times. The availability of such

therapy and the skill to utilize it are limiting factors in its general application.

Intestinal lavage is justified and of great help in those cases with marked azotemia and hyperkalemia, in which the potassium intoxication may be fatal if not corrected. In such cases, intestinal lavage will reduce the potassium and protein levels.

The use of decapsulation and sympathetic nerve blocks has not been productive of improvement. Capsulotomy may itself produce a fatal outcome in a patient in which the outlook is already hazardous. We know of no reports in which sympathetic blocks have influenced the course of patients on which it was applied.

I hope this short summary of this condition will interest internists and general practitioners, as well as obstetricians, to the point of stimulating the prompt treatment of hemorrhagic accidents in pregnancy and labor, and thereby promote the adoption of adequate precautions and treatment by all those in charge of the safe conduct of labor and delivery.

PULSELESS DISEASE

PRELIMINARY REPORT

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Santurce, P. R.

Pulseless disease is a rare clinical entity. It has been reported mainly in the Japanese Medical literature, but a few reports have also appeared from Europe and the United States. Our case is apparently the first one to be reported in Puerto Rico and as far as we have been able to determine, is the youngest known case in the medical literature. We had the opportunity of doing antemortem and post-mortem arteriographic studies in addition to a complete pathologic work up.

CASE REPORT

The patient, a 5 year-old girl was admitted to the Pediatric Service of the San Juan City Hospital on the 13th. of July 1954.

She had been well till 1952 when she began to have repeated "sore throats". In December 1953 she had an episode of fever, migratory polyarthritis and nodules over the anterior tibial area. She was treated by her family physician and after three month of therapy had some improvement, but never had been completely well since then. Two days previous to admission she developed fever, headache, precordial pain, and shortness of breath; and she was referred to us for hospitalization.

On admission she had a temperature of 101ºF, a pulse of 200 per minute, and a respiratory rate of 48 per minute. She was in acute Congestive Heart failure. The heart was enlarged to percussion, had gallop rhythm and a systolic murmur could be heard around the 3rd. interspace to the left sternum. The blood pressure and the pulse could not be obtained in the upper extremities, yet the carotids and the femorals could be easily palpated and there was hypertension in the lower extremities. The blood pressure in the right leg being 180/120 and in the left 190/120. She was digitalized, antibiotics were given and she received other supportive therapy. She responded well to this regime and in about one week she was fully compensated. The pulse rate was around 100 per minute, the respiratory rate around 25 per minute, and the systolic murmur had disappeared.

Blood serology and repeated urinalysis were negative. A leucocytosis was present which was occasionally associated with an eosinophilia. The non-protein nitrogen was normal. Sedimentation rate was 48 Mm per hour. Alpha and Beta hemolytic streptococci were cultured from the throat. Electrocardiograms were

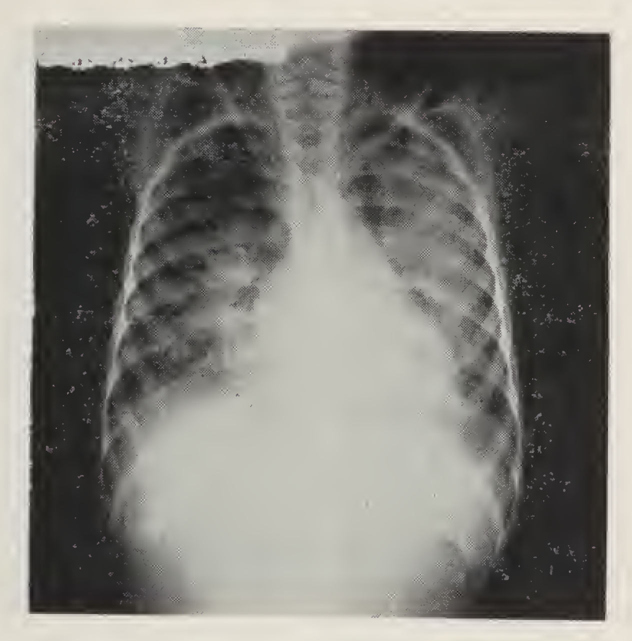


Fig. 1 — Chest film on admission reveals transverse enlargement of the heart with pulmonary congestion.

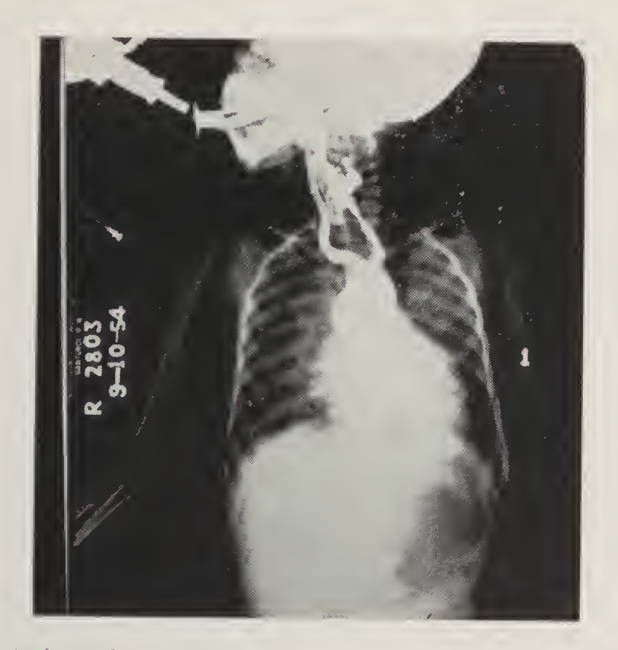


Fig. 2 — Arteriography injecting the left common carotid in a retrogade fashion reveals a normal thoracic aorta with filling of the coronary branch. The innominate and rt. c. carotid filled normally but not the right subclavian br. Only a very short pedicle filled corresponding to the left subclavian. Extensive collateral circulation evident around both shoulders.

normal. X-ray studies of the heart only showed a slight enlargement in the transverse diameter. Oscillometrics readings of the upper extremities gave an index of zero, with a normal index in the lower extremities. Our ophthalmologist saw the case in consultation, and reported that: "The eye grounds showed definite narrowing of the retinal arteries which were also very straight as seen in adults which have early essential hypertension". A muscle biopsy was also done which was negative.

On the 28th. hospital day, after the patient was in fairly good condition, a retrograde arteriography was done through the left carotid artery. She tolerated the procedure fairly well, but two days later she developed respiratory difficulties for which she had to be reintubated; she became decompensated and had to be redigitalized and on the 34th. hospital day she developed generalized convulsions during which she died.

An autopsy was done, and at this time the department of Radiology did postmortem angiographic studies by injection of a barium solution into the arteries.

DISCUSSION

Pulseless disease was described for the first time, in the Japanese medical literature in 1908 by Takayasu; and since then there have been reported about seventy more cases; fifty eight of these from Japan, two from the United tates and the rest from Europe. Most of the cases occur in young females in about a 10 to 1 proportion. The youngest reported so far was an 11 year old girl.

The disease is a chronic one producing general symptoms and accompanied by an increase in sedimentation rate and an increase in the globulin fraction of the serum proteins. The main disturbance is narrowing or complete occlusion of the large arteries that come off the arch of the aorta; and the specific signs and symptoms depend on the vessels that are affected. In our case the vessels mainly affected were both subclavian arteries so that no radial, brachial, axillary or subclavian pulse could be felt bilaterally. The carotids were pulsating so that this case did not show the part of the clinical picture of pulseless disease which is secondary to obliteration of the carotids and which can produce signs of carotid sinus sensitivity, cerebral ischemia and eye changes.

The absence of pulse in the upper extremities associated with the hypertension in the lower extremities, plus the evidence of collateral circulation around the upper part of the chest, even including in some cases notching of the ribs, is why this disease has been called sometimes "reverse coarctation of the aorta".

The retrograde arteriography showed that the arch of the



Fig. 3 — Late film of arteriographic examination discloses a normal abdominal aorta to its bifurcation with remarkable diminution in caliber of the right renal artery in comparison with the left one.



Fig. 4 — Post mortem injection of right axillary artery reveals a very narrow vessel interrupted at the subclavian level and with extensive collaterals around the shoulder and anterior chest wall. Results were similar in the left side.

aorta, the carotids and the descending aorta could be well visualized; but that the subclavian arteries on both sides did not show any dye. Both carotid arteries were seen to be coming out of a common trunk, which is a relatively frequent anomaly among the vessels coming out of the aortic arch. In the left side of this common trunk there was a small area filled with dye which was interpreted as the origin of the obliterated left subclavian artery. On the R carotid there was also seen a small outward bulge which was interpreted as the origin of the obliterated right subclavian artery. The left renal artery and the arterial blood supply of the left kidney were very well outlined, while the right renal artery could not be well visualized. Another point of interest was the filling by the dye of the left coronary artery and its branches.

A system of collateral circulation, similar to the one seen in coartation of the aorta of the adult type, could be seen well. The main anatomatic channels were located around the apex of the thoracic cavity, around the shoulder girdle and through the internal mammary artery. Specially prominent were the upper intercostal arteries which showed the tortuous loops that have been described in coarctation of the aorta.

The post-mortem angiography reproduced practically the same radiologic picture that we had obtained with antemortem angiography. A retrograde arteriography done in both brachial arteries further demonstrated the occlusion of the subclavian arteries up to the beginning of the axillary arteries. The collateral circulation was again demonstrated. Cerebral angiography also done at this time showed a normal cerebral arterial pattern.

The primary pathological finding in this case was the fact that together with the giant cell arteritis that was found in some of the great vessels, there was also evidence of generalized Periarteritis Nodosa. Both of these disease entities belong to the same group of diseases of "allergic-hyperergic" origin and the fact that they occurred together, strengthens the ling between the two diseases. At the beginning of the study of the case the pathologist thought that he might explain the arteritis on the basis of periarteritis nodosa of the vasa vasorum of the great vessels, but no evidence was found of this, and instead the typical pathological picture of granulomatous Giant Cell Arteritis was found.

Another feature of interest was the demonstration that the pathologic process was not restricted, the great vessels coming out of the aortic arch, but that in our case there was also definite narrowing of the right second intercostal artery, the right renal artery and the left iliac artery. Secondary to the narrowing of the right renal artery there was atrophy of the right kidney which can be a factor in the production of the hypertension in this case.



Fig. 5 — Post mortem injection of barium with the aortic arch clamped at its base and just below the ligamentum arteriosus. Again reveals normal filling of the innominate and both carotids with non filling of the right subclavian and only of a short pedicle corresponding to the base of the left subclavian.



Fig. 6 — Filling of the rest of the aorta after removal of the distal clamp during post mortem barium injection.



Fig. 7 — Lateral view of No. 6.



Fig. 8 — Filling of the cerebral circulation including both carotids and vertebraes during the barium injection.

SUMMARY

A case of Pulseless disease has been presented. The main clinical features were the absence of pulse in the upper extremities together with hypertension in the lower extremities. Antemortem and post-mortem arteriography correlated very closely. Pathological examination disclosed the presence of both Periarteritis nodosa and Giant Cell arteritis.

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PALABRAS PRONUNCIADAS POR EL DR. J. BASORA DEFILLO DURANTE LA ASAMBLEA ANUAL DE LA ASOCIACION DE DUEÑOS DE FARMACIAS

Sr. Presidente, miembros de la Asociación de Dueños de Farmacia, Señoras y Señores:

A nombre del presidente de la Asociación Médica de Puerto Rico les saludo cordialmente y les deseo mucho éxito en sus deliberaciones. La Asociación Médica agradece mucho la distinción y el honor de estar representada en esta Asamblea.

Siempre, he considerado a la clase farmacéutica dentro de la misma familia médica. Ambos están íntimamente relacionados. Al médico se le encarga la atención del enfermo; al farmacéutico se le confía el despacho de la medicina prescrita por el médico. Ambos tienen un interés común: el mejorar y proteger la salud del ser humano.

El farmacéutico o su ayudante de farmacia juegan un papel muy importante en el desarrollo de buenas relaciones humanas entre el médico, el farmacéutico y el público. La farmacia es un gran centro de relaciones públicas y de educación en materia de salud, donde el público puede ilustrarse. Tomemos por ejemplo el charlatanismo. Aun en este siglo, hay un sector de nuestro pueblo que vive en la ignorancia, que es apático para consigo mismo, permitiendo que persistan síntomas que pueden ser signos de enfermedades peligrosas. La ignorancia lo hace presa del charlatanismo que todavía abunda en nuestro medio. Si pasamos por muchos de los barrios de la ciudad capital y de las ciudades de la isla, y aún en muchas de sus avenidas, veremos los grandes cartelones de llamadas consultas de clarividentes, medium espiritistas, y doctores en ciencias ocultas; veremos también cartelones llamativos de quiroprácticos indicando poder curar todas las enfermedades por medio de manipulaciones, sin usar medicinas ni inyecciones. Todas aquellas personas sensatas e inteligentes que vean estos tendrán que exclamar: "Eso no es otra cosa que un engaño para el pueblo". La clase médica ha combatido estas prácticas, pero le ha sido imposible eliminarlas en total pues muchas de estas personas siguen siendo patrocinadas por el público.

El farmacéutico debe ayudar a hacer desaparecer esta clase de amenaza a la salud del pueblo. Es bien sabido como en un gran por ciento de farmacias se despachan recetas no-éticas que son indicadas por estos charlatanes arriba mencionados. Recetas tales como: "uña de la gran bestia", "aceite de culebra", y mejurjes prescritos por personas inescrupulosas.

Recientemente vino a mis manos un folleto titulado "El

Poder de los Reflejos" por el Profesor E. Vielle. Sin duda alguna, el mote de Profesor E. Vielle es un pseudónimo. Este folleto, escrito para inculcar en la mente del pueblo una serie de confusiones, es una obra de charlatanería. No sé exactamente la circulación que ha tenido el librito. Su contenido es una serie de escritos donde se le dice al lector como, con una serie de mejurjes llamados reflejos, puede la persona cambiar de suerte, influenciar a otros, curarse y otras falsedades. Si es que estos productos no-éticos son vendidos en alguna farmacia, deben ser eliminados. El farmacéutico que despacha esta clase de compuestos está fomentando el charlatanismo y ayudando a la confusión y a la ignorancia del pueblo. El que fomenta el charlatanismo está insultando la moral cristiana.

El enfermo inculto en cuyas manos cae semejante folleto o lee los anuncios en los periódicos sobre productos no-éticos, o por la radio oye los locutores alabando las maravillas curativas de productos sin valor terapéutico alguno, se siente atraído por un medio cómodo y barato de curarse. Engañándose a sí mismo con detrimento incalculable a su salud o la de sus semejantes. No debe permitirse ni fomentarse por radio, televisión, prensa y menos aún por el farmacéutico o por el médico, el anuncio de supuestos métodos curativos que no responden a la verdad científica.

Muchas personas, por no poder pagar o por no querer pagar al médico, y las más de las veces por negligencia e ignorancia, acuden a la farmacia más cercana para que le receten cuando hay enfermos en su casa. Sabemos que se acostumbra recetar y despachar medicinas en algunas farmacias solamente porque un cliente viene a ese farmacéutico y le relata una serie de síntomas. Esta es una forma incorrecta de recetar, pues para radicar medicación se requiere un diagnóstico y solamente el médico, después de un examen cuidadoso del enfermo, puede hacer un diagnóstico. El encargado de la farmacia tiene aquí una gran oportunidad de instruir al público, indicándole a la persona que así busca una receta el peligro de tal proceder. No debe venderse antibióticos sin previa autorización médica. Hoy en día se abusa de estos productos. Tenemos que darle gracias a Dios por haber iluminado al hombre en el descubrimiento de productos tales como la penicilina, la estreptomicina, aureomicina, cloromicetina, terramicina, isoniacida y otros tantos que están en investigación y que han sido la salvación de tantas vidas humanas. Sin embargo, no debemos caer en el error de usar estos productos innecesariamente y cuando no están indicados. La indicación debe partir del médico. Algunas farmacias también venden barbitúricos sin prescripción y hasta por amistad y confianza con el cliente, acceden a vender narcóticos sin prescripción, fomentando así el uso de ellos, creando hábitos indeseables.

En las farmacias no debe repetirse las recetas de los médicos, si éstas plenamente no indican que pueden repetirse. Es costumbre de muchos clientes el solicitar la repetición de la receta cuando se creen tener la misma enfermedad, sometiéndose a tratamientos innecesarios. Recuerdo hace poco, una señora me llamó alarmadísima pues le había administrado cloromicetina a su hija por varios meses, en tratamiento de un catarro que no le cedía. El antibiótico no se le había prescrito para catarro y sí para una infección faríngea, pero ella sin embargo lo siguió usando por su cuenta. En esos días se había indicado la posibilidad de que este producto produjera anemia aplástica. Esa fué una buena oportunidad mía para indicarle los peligros de la autoterapia. El farmacéutico debe despachar la receta del médico según éste la prescribe y si hay duda preguntar al médico. El que os habla ha pasado por algunas experiencias desagradables con relación a recetas prescriptas por él. Puedo citar los siguientes dos casos: Estando yo en el Hospital de Distrito de Bayamón se le indicó Toxoide tetánico a un niño. El encargado de despacharla le dió Suero antitetánico. En otra ocasión se recetó una ampolleta de globulina inmune y al cliente se le vendió insulina globulina. De haberse administrado esta insulina, las consecuencias habrían sido fatales.

Hay muchos productos de patentes no-éticos y de dudosa eficacia terapéutica que hoy día se encuentran en los anaqueles de muchas farmacias. Estos productos no los recetan los médicos éticos, pero son vendidos por algunas farmacias y son empleados por el público pues se le hace una campaña continua de anuncio por la radio y la prensa. Tibisán, Elixir de Leonardi, Tónico de San Lázaro son nombres de algunos compuestos que vienen a mi mente. El público gasta miles y miles de dólares en estos productos, dinero que va a llenar las arcas de los manufactureros de estos productos que se compran sin mediar receta médica.

He visto con agrado en muchas farmacias un rótulo donde dice: "En esta farmacia no se ponen inyecciones". Felicito a esos farmacéuticos y dueños de farmacia que así actúan. Las inyecciones debe administrarlas el médico, el practicante, o la enfermera graduada bajo supervisión del médico o por indicación de éste. Muchas personas que acostumbran poner inyecciones no se percatan del peligro de tal acción. A menos que se autorice por el médico no se deben administrar inyecciones, más aun si no han sido prescritas previamente.

Hay varias casas manufactureras de productos farmacéuticos que llevan campañas encaminadas a educar al pueblo en relación con su salud, demostrando el interés que debe tenerse por guiar

correctamente al pueblo. La casa Parke Davis publica una serie de anuncios éticos en las principales revistas de Estados Unidos tales como Life, Colliers, Today's Health y otros. En estos anuncios el tema principal es la necesidad de consultar al médico cuando el individuo se enferma. S.K.F. ha filmado varias películas de carácter educativo en relación con temas tales como Corazón, Cáncer, Artritis, Salud Pública, Obesidad, y otros. Estas películas tomadas bajo los auspicios de S.K.F. son respaldadas por la Asociación Médica Americana y teleradiadas a través de cadenas importantes en la televisión en el Continente. Al igual que estos dos ejemplos, están también la Casa Squibb, Abbott, Mead Johnson, Lederle, Pfizer, Sharp & Dohme y otros, que publican también anuncios éticos y cuyos productos son también éticos, sin indicación que pueda inducir a la autoterapia.

El farmacéutico puede ayudar al público estimulando en la mente del pueblo la necesidad de que toda persona enferma vea al médico cuando de su salud se trata. Se debe fomentar la idea del médico de familia. Un médico para cada familia es una idea muy acertada. En reciente discurso el ex-Presidente de la Asociación Médica Americana, Dr. McCormick expresó sus ideas sobre este particular, instando al médico, al farmacéutico y al público a endosar y fomentar esta idea. La casa Parke Davis trae recientemente en su publicación "Notas Terapéuticas" la reproducción de un anuncio donde el tema principal es "El Médico de Familia". En la Asociación Médica de Puerto Rico los ex-presidentes, Dres. Sanjurjo, Hernández Morales y otros también han hablado sobre la importancia del médico de familia. Ustedes como personas influyentes en la comunidad, personas que vienen en contacto con el pueblo, están en mejor posición que nadie para ser vehículo de distribución de estas ideas y prácticas de salud. Ustedes pueden ayudar a ilustrar al pueblo a hacerle comprender el daño que se infligen con la autoterapia.

El farmacéutico y el médico en una mutua cooperación pueden cambiar la actitud del ciudadano para cuidar propiamente de su salud. Estamos todos obligados a educar al pueblo, a sacarlo de la ignorancia. Debemos anteponer nuestros sentimientos humanos a los del negocio cuando de la salud se trata. El engaño es peligroso.

Repito mis saludos y felicitaciones a los actuales dirigentes y a los miembros de la Sociedad y le deseo éxito a la nueva directiva que estáis a punto de instalar.

THE DISABILITY PROVISION OF THE FEDERAL OLD-AGE AND SURVIVORS INSURANCE PROGRAM AS IT IS APPLIED TO PUERTO RICO

One of the recent amendments to the Social Security Act preserves the old-age and survivors insurance rights of individuals who have been totally disabled for an extended period before attaining age 65. Puerto Rican physicians will find information about the new provision useful in their practice because disabled patients may request help in establishing eligibility for the "freeze".

Increasing efforts are being made to restore more of our disabled persons to gainful activity. In 1954 Congress improved and expanded the scope of the Vocational Rehabilitation Act, and made available additional funds under the Hill-Burton Act for the construction of rehabilitation facilities. The "disability freeze" amendment to the old-age and survivors insurance provisions of the Social Security Act will serve also to stimulate vocational rehabilitation activities throughout the nation. The Bureau of Old-Age and Survivors Insurance, a part of the Social Security Administration in the Department of Health, Education, and Welfare, has responsibility for the operation of the Federal old-age and survivors insurance program. The officials of this Bureau have asked the Nation's medical profession for cooperation and counsel in the formulation of sound standards and the application of them in making determinations of disability for purposes of this program.

Under the Old-Age and Survivors Insurance program, wage earners, their employers, and self-employed persons contribute to a fund from which wage earners and self-employed persons may be paid monthly old-age insurance benefits upon retirement after age 65, and, from which their survivors may be paid monthly benefits.

The "disability freeze" provisions bears some resemblance to the waiver-of-premium clause in commercial life insurance policies. It does not provide cash benefits during a period of disability; it simply enables an individual to preserve his rights to old-age and survivors insurance benefits during a period when he is unable to work because of total disability which has existed for some time and is expected to be of prolonged duration. It provides for the use of the skills of professional persons in State and local agencies to get the facts about an applicant's physical or mental impairment. It also directs the Department of Health, Education, and Welfare to refer the disabled applicant to a State agency for possible vocational rehabilitation services.

For a person to become entitled to old-age insurance benefits,

or for his family to become entitled to survivors' benefits in case of his death, he must have engaged in work covered by the program for a specified length of time. The amount of monthly benefits is based upon his "average monthly earnings" in such work. Before the enactment of the disability freeze, periods of low or no earnings, such as those caused by disability, reduced his average monthly wage and resulted in lesser monthly benefit amounts. It was even possible to lose eligibility to benefits because of prolonged absence from covered work. The new provision preserves a disabled person's right to full benefits by allowing the exclusion of a period of extended disability in the computation of his benefit amount.

The term "disability" is statutorily defined as:
"*** inability to engage in any substantial gainful activity by reason of any medically determinable physical or mental impairment which can be expected to result in death or to be of long-continued and indefinite duration, or blindness."

The only impairment which is specifically defined is "blindness". It is defined as central visual acuity of 5/200 or less in the better eye with the use of a correcting lens. An eye in which the visual field is reduced to 5º or less concentric contraction is considered as having a central visual acuity of 5/200 or less. (An individual with a visual impairment which does not meet this definition may, however, be considered and may be found eligible under the general definition.) A person whose sight is impaired to the extent defined in the law is deemed blind regardless of whether he is able to engage in gainful activity. The determination of whether an individual's impairment may be deemed to be a "disability" must be made upon consideration of the medical evidence and by application of medically approved standards of disability.

In general, determinations of disability will be made by a State vocational rehabilitation or other appropriate State agency, under an agreement entered into with the Secretary of Health, Education, and Welfare. The State agency will be reimbursed for the necessary costs of making such determinations. In Puerto Rico, the determinations of disability will be made by the Vocational Rehabilitation Division.

A large number of freeze applications are expected in the early months of 1955 because persons with sufficient covered earnings who are currently disabled may establish periods of disability retroactively to the earliest date they were disabled and could meet the covered work requirements for a period of disability.

The Bureau has enlisted the counsel of a Medical Advisory Committee whose members will be representative of the various fields

of the medical profession. This committee will assist in the development of policies, standards, and procedures for the procurement and evaluation of medical evidence in accordance with sound medical practice.

In formulating standards and guides for the evaluation of disability, the Bureau of Old-age and Survivors Insurance and the Medical Advisory Committee will have the benefit of experience acquired by professional personnel in the administration of Federal and State disability programs such as vocational rehabilitation, the veterans' programs, workmen's compensation, aid to the permanently and totally disabled, and other disability retirement programs.

The medical aspects of a disability determination will be based upon medical evidence furnished from either of two sources:

1. The person may ask his physician to send an abstract concerning his impairment to the office handling the person's application for a disability determination. A brief form which has been prepared for this purpose will provide space for medical history, clinical findings, and diagnosis of the impairment. In some cases, it may be necessary for the disabled person to have a current medical examination in order to meet the requirement that he furnish medical evidence to support his application; or

2. The disabled person may arrange to have submitted in his behalf a summary of an existing medical record in a hospital or other institution or agency.

If the medical evidence submitted by the applicant is not acceptable or is inconclusive, a current medical examination may be authorized. Arrangements for such examinations will ordinarily be made by the State agency with a local physician, as is now done when the agency secures an examination for its own purposes. Payment for such examinations will be made by the State agency which in turn, will be reimbursed by the Bureau of Old-Age and Survivors Insurance.

To qualify for a disability determination, a person must have worked in employment covered by the Social Security Act for a substantial period prior to the onset of his impairment. Briefly, he must be credited with earnings for at least 5 years out of the last 10 years, including at least 1½ years out of the last 3 years immediately before the onset of his disability.

A person's earnings record cannot be frozen until he has been disabled for 6 months and it appears that he will suffer prolonged disability or death. In addition, he must be still disabled when he files his application for the disability determination. A period of disability ends when the disability ceases or he attains age 65.

A person who files an application before July 1, 1957, for a

"freeze" may establish a period of disability which extends retroactively to the time when he could have first met the eligibility requirements. A person who is now receiving old-age insurance benefits and who was totally disabled before age 65 may have the amount of his monthly benefits increased by filing a valid application for a "freeze". However, no benefit amounts can be increased for months before July 1955, and the applicant must be living on July 1, 1955, for his application to be valid.

An application to establish a period of disability for Old-Age and Survivors Insurance purposes should be filed by the worker in the nearest social security district office. There are 5 district offices serving residents in Puerto Rico. These are located in Arecibo, Mayagüez, Caguas, Ponce and San Juan. Itinerant service in other communities is provided by each of these offices, and a representative will call on persons who may be confined to their homes. Additional information concerning the disability "freeze" program can be obtained from any of the above district offices.



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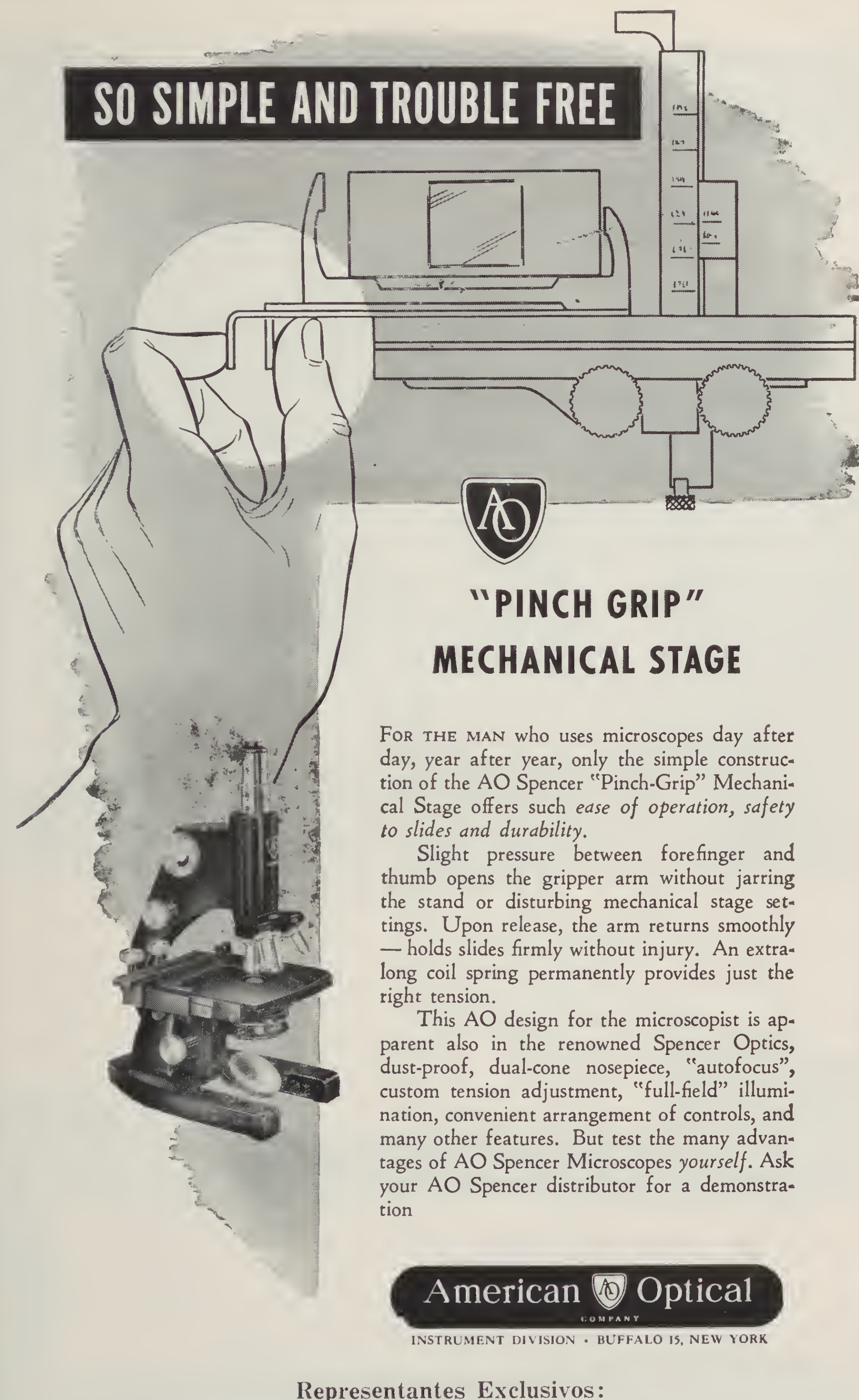
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BOLETIN

DE LA ASOCIACION MEDICA DE PUERTO RICO

VOL. 47 MARZO, 1955 No. 3

CLINICAL EXPERIENCE WITH MYLERAN IN THE TREATMENT OF LEUKEMIA*

*RAMON M. SUÁREZ, RAMON M. SUÁREZ JR., JUAN SABATER and ROBERTO BUSÓ***

(With the technical assistance of Mrs. Sara Torres de Olavarrieta and Mrs. Jutta Munchow de Graulau.)

Localized roentgen irradiation and internal total body irradiation with radioactive phosphorus P_{32}, have been considered the most effective treatments of chronic myelocytic leukemia. Beneficial results have also been obtained with the use of nitrogen mustard, triethylene melamine (TEM), urethane and total body roentgen irradiation.[1] A new colchicum alkaloid known as Demecolcin Ciba[2] has also been found to have cytostatic effects in myelogenous leukemia.

Recent studies conducted in Europe and in the U.S. seem to indicate that a new drug, a sulfonic acid ester, originally prepared at the Chester Beatly Research Institute in London and known as G.T.41 or Myleran has some value in the treatment of chronic myeloid leukemia. Its chemical name is 1,4-Di (methanesulphonoxy)butane and its formula, CH_3-SO_2-O-(CH_2)4-O-SO_2-CH_3.

English investigators found the drug to be active against the Walker rat carcinoma 256 and that it produced marked depression of granulopoiesis, not only in rat, but also in man. Its depressive effect seemed to be selective on the granulocytes. In contrast to roentgen rays and the amine mustards lymphopoiesis was apparently not affected.

The early clinical trials by Galton[3] and Haddow and Timmis[4] in Britain showed a favorable effect of Myleran upon patients with chronic myelogenous leukemia. They reported a rise in hemo-

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* Presented at the annual meeting of the Medical Association of Puerto Rico on December 9, 1954.

** From the staff of Fundación de Investigaciones Clínicas and Hospital Mimiya, Santurce, Puerto Rico.

The drug was supplied by Dr. Donald S. Searle, Medical Director of Burroughs Wellcome & Co. (U.S.A.) Inc., of Tuckahoe, N.Y.

globin, a decrease in the number of leucocytes, a selective reduction or even disappearance of immature myeloid cells from the blood, a reduction of the cellularity of the bone marrow, a diminution in the size of the spleen, and marked subjective improvement.

The results were soon confirmed by Bollag[5] in Europe, and by Wintrobe et al,[6] Burchenal,[7] and Petrakis and co-workers[8] in the U.S. The most recent contributions were those of W. C. Levin[9] and J. Louis,[10] with their respective collaborators, presented at the 27th. Annual Meeting of the Central Society for clinical Research held in Chicago on October 29 and 30 this year.

There seems to be general agreement as to the efficacy of this new chemotherapeutic agent in the management of chronic myeloid leukemia, but important points as to optimal therapeutic and maintenance dosage, duration of treatment, effect of repeated courses, character and duration of remissions, the effect on the bone marrow and specially on platelet formation, the possibility of inducing acute myeloblastic crises and a few other problems remain to be determined.

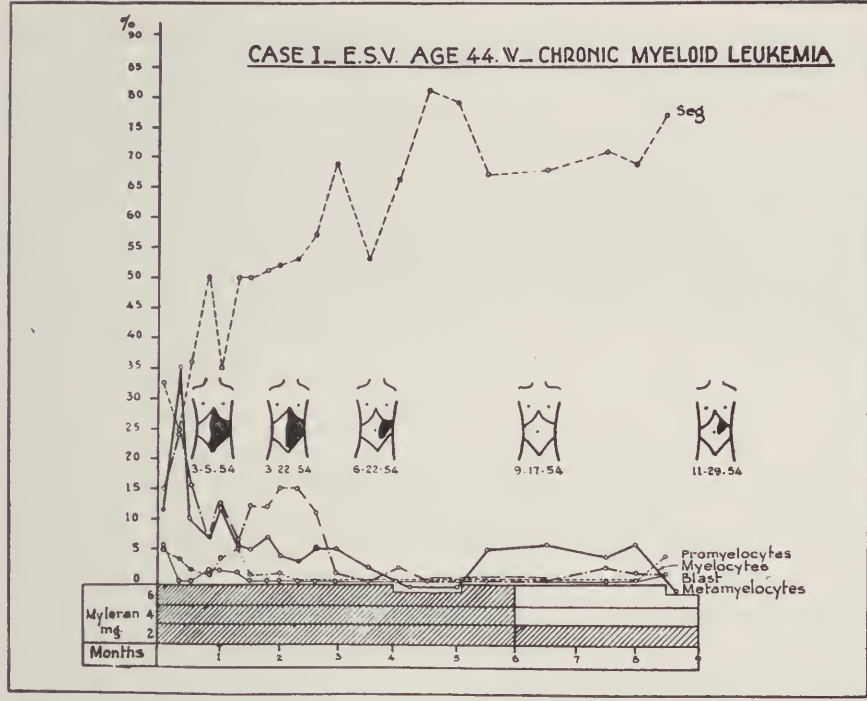
Our experience with this chemotherapeutic agent is limited to three cases of chronic myelocytic leukemia, and one case of subacute monocytic leukemia.

CASE 1

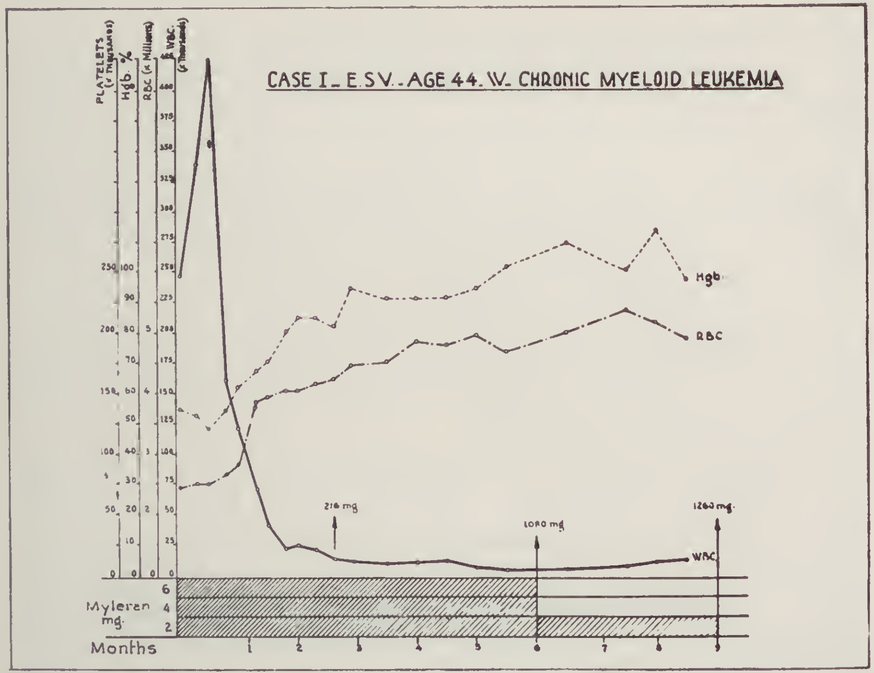
E. S. V. — A 44 year old white widower, employed as male nurse at the Insular Tuberculosis Sanatorium was admitted because of pain on the left side of the abdomen of 2 years duration. A diagnosis of myelogenous leukemia had been established at the Bayamón District Hospital in 1952. He had received numerous blood transfusions; X-ray therapy to the spleen was given in July, 1952, and repeated 6 months later.

On physical examination, we found an anemic man, weighing 118 pounds, height 63 inches, blood pressure 100/70. Eyes showed normally reacting pupils and normal fundi. He was wearing well fitting dentures. There was no lymphadenopathy, no hemorrhagic tendency, no fever. The spleen was enormously enlarged, filling up the entire left side of the abdomen and part of the right side, and its lower pole lying below the brim of the pelvis could not be palpated.

The peripheral blood showed 2.45 million red blood cells, 7.9 gm. (55.3%) hemoglobin and 244,000 leucocytes per c.mm. Platelets were not diminished. The differential count confirmed the diagnosis of myelogenous leukemia. There were 5.5% myeloblasts, 5% pre-myelocytes, 13.5% neutrophilic myelocytes and 11.5% me-



Effect of Myleran on the size of the spleen and on the differential count of the leucocytes. There was a decrease in blasts and more immature forms and an increase in polymorphonuclear leucocytes.



Effect of Myleran on leucocyte count, erythrocytes and hemoglobin. The daily and total doses are given.

tamyelocytes. Neutrophilic polynuclear cells were 32.5%, Stabs 26.5%, basocytes 2.5% and lymphocytes were only 2.5%.

Myleran in doses of 2 mg. three times a day was administered. No more blood transfusions were given. The improvement, both subjective and hematologic was slow but definite and progressive. The patient experienced a sense of well being and felt stronger by the second week of treatment. A sharp increase in the total number of leucocytes (from 244,000 to 423,000 per c.mm.) with a slight drop in hemoglobin (from 7.6 gm. to 6.9 gm.) was observed on the 14th day of treatment, but 7 days later a definite improvement in the blood picture took place. At this time, red blood cells were 2.64 million, hemoglobin 7.9 gm. (55.3%) and leucocytes 160,750 per c.mm. On the 36th. day, when a total of 216 mg. of Myleran had been administered, the leucocytes had decreased to 22,000 and the red blood cells had increased to 4.02 million and hemoglobin to 11.6 gm. (80.1%). By the 6th. month of treatment, the patient's peripheral blood appeared normal: the total leucocyte count was 5,000 per c.mm.; polymorphonuclear neutrocytes were 68%, basocytes 4%, and lymphocytes had increased to 10%, and no myelocytes were observed. The erythrocytes had increased to 5.36 million and hemoglobin to 14.5 gm. (100%). Platelets were 154,000 per c.mm. At this time the patient was put on a maintenance dose of one tablet (2 mg.) Myleran daily.

The most striking objective evidence of the effect of Myleran was that observed on the spleen. The enormous organ began to diminish in size by the third week of treatment, and by the sixth month it could not be palpated by any one of several examiners. One month after the dose of myleran was diminished from 6 to 2 mg. daily, the spleen again became barely palpable on deep inspiration, and two months later it was easily palpable, reaching 2 inches below the left costal border.

The selective effect of Myleran on the granulocytes was evidenced by an early depression of the more immature cells. The blast forms and the promyelocytes disappearing first from the peripheral blood, followed by a decrease in the number and final disappearance of the myelocytes.

It may be interesting to state that even at the time when the peripheral blood could be considered normal, the bone marrow, despite marked improvement in the number of normoblasts and erythroblasts, and a definite decrease in the number of myeloblasts and myelocytes and apparently less hyperplasia or cellularity, the picture was still compatible with the diagnosis of chronic myelocytic leukemia. It is also interesting to mention the fact that, despite the sense of well being, the increased capacity to work, and the

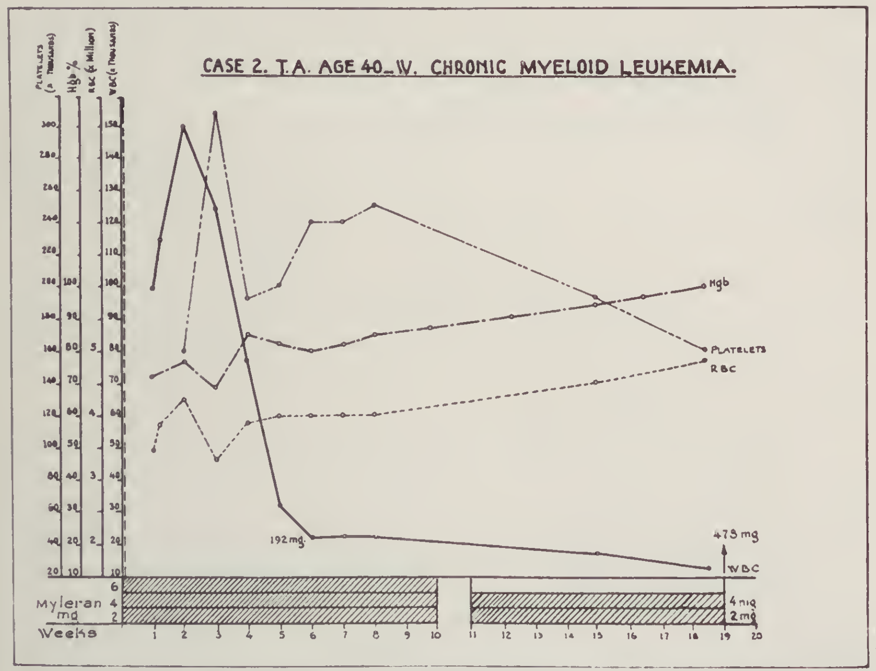
apparently healthy appearance, and the normal basal metabolism (+9%), no increase in weight was observed during the period of observation, which has lasted nine months.

The last bone marrow study performed on Nov. 29, 1954, showed 1% reticulum cells, 2.5% myeloblasts, 4.5% pre-myelocytes, 9% neutrophilic myelocytes, 44% metamyelocytes, 14.5% neutrophiles, 3% eosinophiles, and 12% band forms. Platelets were 139,000. There was no hypoplasia and no thrombocytopenia. We were justified, therefore, in increasing the dose of Myleran again to 6 mg. daily, as we did on Nov. 30.

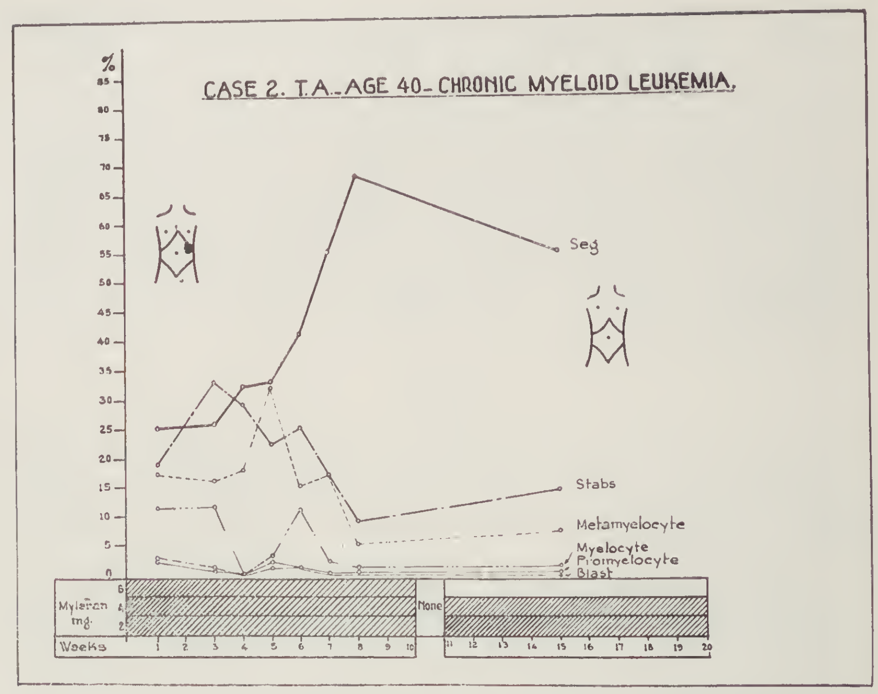
CASE 2

T. A. — A 40 year-old white Veteran's Bureau beneficiary was admitted on July 16, 1954, because of left mid-abdominal pain, dull in character, of 3 months duration. He had lost 10 pounds of body weight.

Physical examination was negative, except for slight splenic enlargement. Studies of the peripheral blood and of the sternal marrow obtained by aspiration confirmed the diagnosis of chronic myelocytic leukemia and Myleran, 2 mg. tablets, three times a day, were given, beginning on July 23, 1954.



The effect of myleran on the leucocytes, erythrocytes and hemoglobin. Daily doses administered and total dosage.



Effect of Myleran on the spleen and on the leucocytes. A diminution of the more immature forms and an increase of the segmented leucocytes.

The blood picture on entry showed a red blood cell count of 3.43 million, hemoglobin of 10.4 gm. (72.6%), leucocytes 114,500 per c.mm. and platelets 160,000 per c.mm. The peripheral blood picture was indistinguishable from that of a normal bone marrow. There were 2% myeloblasts, 2.6% pre-myelocytes and 11.4% neutrophilic myelocytes. In the marrow, the granulocytic series made up 97% of the nucleated elements.

The leucocytes began to decrease by the third week of treatment, and on Aug. 25, 1954, thirty two days after the institution of therapy, the leucocytes had decreased to 22,500, platelets had increased to 240,000, red blood cells were 4.01 million, and hemoglobin 11.6 gm. (80.1%).

Clinical and hematologic improvement continued. The spleen was no longer palpable. The patient was put on a maintenance dose of 4 mg. daily, and was discharged on Sept. 8, 1954, to be followed at the out-patient clinic. His last blood picture on Nov. 22, 1954 appeared practically normal. Red blood cells were 4.86 million, hemoglobin 14.5 gm. (100%), platelets 160,000, and leucocytes 12,500 per c.mm. No myeloblasts and only 2% neutrophilic myelocytes were seen in the blood smear. A total dose of 47.8 mg. of Myleran had been given.

The bone marrow picture had shown slight improvement with somewhat less hyperplasia. The granulocytic series had decreased from 97% to 91%, and the erythrocytic series had increased from 3% to 8.8%, but the picture remained consistent with the established diagnosis of myelocytic leukemia.

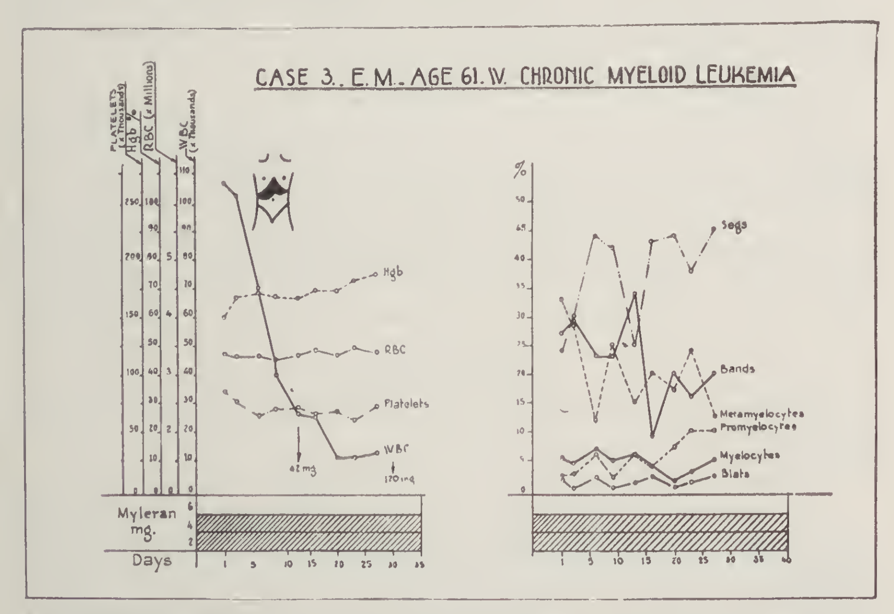
He is, at present, receiving a maintenance dose of 2 mg. daily.

CASE 3

E.M. — A 61-year old white man was referred to us by Dr. R. Mejía Ruiz, from Humacao, P. R., who had established a diagnosis of myelocytic leukemia. The patient had complained of bone and joint pains, weakness and palpitations, and because of persistent hematuria of 4 months duration, he had been posted for cystoscopy at another hospital.

Physical examinations revealed a pale, blue-eyed, afebrile man in no discomfort, weighing 123 pounds, showing spongy infected gums, small discrete inguinal glands, and an enormous liver, reaching 4 inches below the costal border at the mid-clavicular line. Both the right and left lobes were enlarged. The spleen could not be palpated. There was gross hematuria and urinalysis on entry showed specific gravity of 1,019, albumin ++++, negative sugar, many white blood cells and loaded with red blood cells.

The peripheral blood showed the following findings: erythrocytes 3.3 million, hemoglobin 9.7 gm. (76.3%), leucocytes 102,000



No significant effect on the size of the liver was observed despite the marked decrease in the number of leucocytes with only 4 mg. daily.

per c.mm., and platelets 76,000. Bone marrow studies confirmed the diagnosis of chronic myelocytic leukemia.

Because of the low platelet count, and because thrombocytopenia has been reported as a complication of Myleran therapy, we gave this patient only 4 mg. daily, and kept a careful watch on the platelets.

Thirteen days later, when only 52 mg. of the drug had been given, the leucocytes had decreased to 26,000 per c.mm., but the red blood cells, the hemoglobin, as well as the platelets, remained unaffected. The liver seemed to be about one inch smaller. At this time, the spleen was barely palpable, and only microscopic hematuria persisted.

On the 34th hospital day, the patient was feeling well and stronger. The urine showed only 4-5 red blood cells per high power field, from 3 to 35 leucocytes, and ++ albumin. The weight had increased to 128 pounds. Hemoglobin was 10 gm. (69.8%) erythrocytes 3.41 million, platelets 72,000 and leucocytes 15,950 per c.mm., blood uric acid 6.6 mg. and BMR +9%. The bone marrow showed practically no change.

A few days later the patient was discharged, to be followed at home, while Myleran therapy in dose of 4 mg. daily is continued.

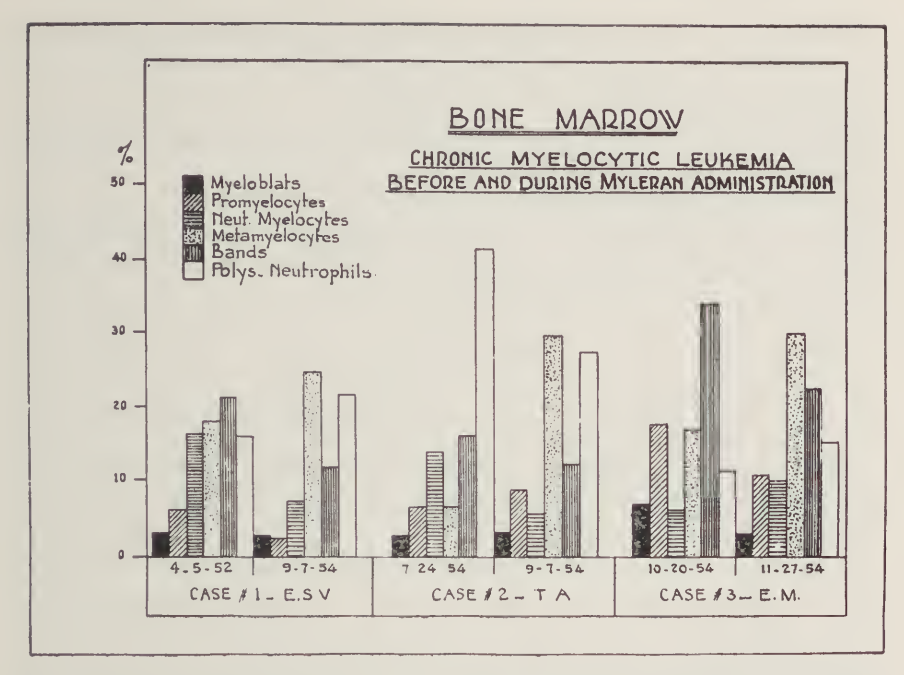
DISCUSSION

Our studies and observations confirm the experience of British and American investigators on the efficacy of Myleran in the treatment of chronic myelocytic leukemia.

The drug has been found ineffective in the acute forms of myelocytic leukemia, and in both acute and chronic forms of lymphocytic leukemia. In the latter disease it is contraindicated.

In a case of subacute monocytic leukemia, which we followed for several months, Myleran induced a decrease in the number of granulocytes and monocytes, and a diminution in the size of the spleen, but the persistence of the anemia required frequent blood transfusions. In a case of multiple myeloma, it was also possible to reduce the number of granulocytes in the peripheral blood, but the number of plasma cells and the course of the disease was not altered.

It has been claimed that Myleran is contraindicated in cases of chronic myelocytic leukemia when the platelet count is less than 100,000 per c.mm. Our case 3, who showed an initial platelet count of 76,000, has been receiving 4 mg. daily for over a month without any evidence of further depression of the thrombocytes and with marked effect on the granulocytes, and definite clinical improve-



The effect on the bone marrow not as marked as that observed in the peripheral blood. Myeloblasts and premyelocytes did not disappear from the marrow.

ment, with disappearance of hematuria, which we had attributed to leukemic infiltration of the kidneys.

Levin and his co-workers reported that 2 of their 6 cases of chronic myelocytic leukemia developed acute myeloblastic transformation, with rapid fatal outcome during Myleran administration. The authors admit that there is need for wider experience and more cases to further evaluate the possibility that this agent may, in some instances, "potentiate the virulence of chronic myelocytic leukemia". We add that in the natural course of chronic myelocytic leukemia, acute myeloblastic transformation may occur spontaneously.

CONCLUSIONS

1—Myleran may induce complete though temporary remissions in the clinical picture and peripheral blood of patients with chronic myelocytic leukemia.

2—Complete marrow remission was not observed.

3—A daily dose of 6 mg. seems adequate treatment in most cases, and a maintenance dose may vary between 2 and 4 mg. daily.

4—No gastro-intestinal disturbances and no side or toxic effects were seen.

5—Whether the best program consists in the administration of repeated courses or the administration of a maintenance dose of the drug remains to be determined.
6—Whether Myleran will prolong or not the life of the patient has not been established as yet.
7—The drug is easily administered and is inexpensive.
8—Although a method of palliation, Myleran is at present the most effective chemotherapeutic agent used in the treatment of chronic myelocytic leukemia.

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MATERNAL MORTALITY SURVEYS IN MINNESOTA*

*A. B. ROSENFIELD, M.D., M.P.H.***

For the past several decades there has been a continuous and significant decrease in maternal mortality rates. This continued reduction is due to several factors, including better undergraduate training of physicians, an increasing number of postgraduate and refresher courses in obstetrics, an increasing percentage of babies born in modern, well-equipped and well-staffed hospitals, widespread use of new chemotherapeutic agents, and early and adequate blood replacement. Public health measures such as education of the public in the importance of early and adequate prenatal and obstetrical care, as well as maternal mortality surveys for the education of physicians and hospital personnel, are other factors in the reduction of maternal mortality. In many states maternal mortality rates are now lower than what was regarded as the irreducible minimum hardly more than a decade ago. In spite of these low rates, well-conducted surveys indicate that many deaths are still preventable.

All state health departments carry on an annual statistical analysis of maternal deaths through their sections of vital statistics and maternal and child health. These studies are based on death certificates that have been shown to be inadequate and frequently misleading, since they show marked differences between the reported and the actual cause of death. Furthermore, many deaths associated with pregnancy are not reported as such.

A recent survey by the American Medical Association revealed that 35 state medical societies had active maternal health or welfare committees. These committees conduct cooperative maternal mortality studies through questionnaires filled out by the attending physician or designated committee members. Less than a dozen states, including Minnesota, carry on a thorough investigation of all maternal deaths by personal visits of trained obstetrician-investigators.

Minnesota's first state-wide maternal mortality survey was conducted from July 1, 1941, through June 30, 1942. It was discontinued during the war because of lack of personnel. The survey was resumed in 1950 and is now in its fifth consecutive year. It is a cooperative study by the Minnesota State Medical Association and the Minnesota Department of Health. A maternal mortality sur-

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* Presented before the Section on Obstetrics and Gynecology, P. R. Medical Association, February 23, 1955.

** Chief, Section of Maternal and Child Health Division of Local Health Services, Minnesota Department of Health.

vey or neonatal study will by its very nature evoke valid criticisms of the handling of a certain number of the deaths under study. No one likes to be criticized, especially by a regulatory agency. Criticism by the physician's own organization, however, is usually more acceptable and thus has greater educational value. While the survey is a joint undertaking, emphasis must be placed on the fact that this study is carried out by the State Medical Association in an effort to reduce preventable deaths by eliminating all possible hazards of pregnancy and childbirth. It therefore has inestimable public relations value for physicians. Trained obstetrician-investigators are selected by the committee and are employed on a part-time basis. Funds for the study are provided by the State Department of Health. Cooperating in the survey are the Department of Obstetrics of the University of Minnesota and the Minnesota Hospital Association.

The survey is conducted by a group of physicians known as the Minnesota Maternal Mortality Committee. Members are selected by the Maternal Health Committee of the State Medical Association and are appointed by the Council of the Association. They serve without remuneration. The committe consists of 11 obstetricians representing all parts of the state in addition to the chief of the Section of Maternal and Child Health of the State Department of Health.

As soon as maternal death certificates are received by the section of Vital Statistics of the Minnesota Department of Health, photostat copies are prepared and forwarded to the Section of Maternal and Child Health. When a certificate or a report of a maternal death is received an appointment is made with the attending physician. The obstetrician-investigator visits the hospital and reviews the hospital chart and nurse notes, the anesthesia record and the pathologist's report. He talks with hospital personnel who came in contact with the patient. He visits the attending physician, obtains the history, including prenatal care, and discusses various aspects of the death, for the completion of a detailed questionnaire. He interviews the consultant, if one had been called in. He is entirely objective and impersonal in his questioning and makes no comment or criticism of the handling of the case. Criticism is the function of the committee.

The investigator then prepares a summary outlining the course of events in chronological order for presentation to the committee at its periodic meeting. The record is anonymous. The committee is told only whether the attending physician was a general practitioner or a specialist, his training, and whether the death occurred in a small or medium-sized rural hospital or in a metropolitan hospital. After thorough discussion by committee members, final

decision is made as to the cause of death, adequacy of care and conduct of the case, contributory and non-contributory factors in regard to prenatal care, labor, delivery, and postpartum care, autopsy findings and their completeness, accuracy of death and birth certificates, preventability of the death, and finally determination of responsibility for the death —whether due to patient, physician, hospital, or disease. A copy of the summary is mailed to the physician and the consultant.

To point out the significant findings of the current survey and to demonstrate the improvement that has occurred in the past decade, a comparison will be made between the results of the survey carried out during the 12-months period from July 1, 1941, to June 30, 1942 inclusive, which will be referred to from now on as the "1941 survey," and the surveys made in 1950, 1951, and 1952 respectively.

Table 1 shows that the 1941 survey included 112 deaths. Ninety-three were obstetrical (maternal) deaths as coded by the National Office of Vital Statistics; 19 were non-obstetrical, that is deaths associated with pregnancy, labor or the puerperium for a period of three months, but not directly due to pregnancy. The 1950 survey included 46 obstetrical and 22 non-obstetrical deaths; the 1951 survey included 39 obstetrical and 18 non-obstetrical; the 1952 survey, 33 obstetrical and 25 non-obstetrical deaths.

It will be noted that while about one-sixth (17%) of deaths included in the 1941 survey were non-obstetrical, more than twice as many (35.5%) in the combined 1950, 1951, and 1952 surveys were non-obstetrical. In fact, in the 1952 survey 43% were non-obstetrical deaths. In other words, with the decreasing number of obstetrical deaths in recent years more interest and effort have been devoted to deaths associated with pregnancy but not directly due to pregnancy. In such deaths interest is centered on the adequacy of prenatal, natal, and post natal care. Incidentally, each year several obstetrical deaths are found in the non-obstetrical group.

There was a marked increase in the number of live births in Minnesota from 1941 through 1952, accompanied by a continuous and marked increase in the number of hospital births as well as a decrease in maternal mortality (Table II). The average number of live births during the 1941 study was 55,293; 76% occurred in hospitals. Ninety-three maternal (obstetrical) deaths occurred with a maternal mortality rate of 1.6 per 1,000 live births. In 1952 there were 79,198 live births; 98.9% occurred in hospitals. There were 28 maternal deaths, a rate of 0.4 per 1,000 live births. There was therefore a 43% increase in the number of live births and a 30%

increase in the number of hospital births, accompanied by a 75% decrease in maternal mortality.

It should be noted that the surveys include all deaths due to or associated with pregnancy or childbirth, as well as those occurring during a period of three months postpartum, regardless of the cause of death. This fact accounts for the larger number of deaths in the study than in the official reports of the Section of Vital Statistics and the U.S. Office of Vital Statistics. The maternal mortality committee includes all deaths, obstetrical and non-obstetrical, and reports a gross or over-all mortality rate. In contrast, the public health definition of a maternal death as accepted by all city and state health departments, the U. S. Office of Vital Statistics, and the World Health Organization is limited to so-called "obstetrical" deaths, that is, puerperal deaths. When deaths considered by the committee as non-obstetrical are excluded, the maternal mortality rate approximates the official Minnesota rate and is comparable with other state rates.

Adequate prenatal care was given in a significantly higher percentage of cases in 1952 than in 1941 - 21 per cent in contrast to 1.9 per cent - but there is room for considerable improvement. Faulty prenatal care that contributed to the patients' deaths was markedly reduced from 53% in 1941 to 11% in 1952 (Table III). Faulty care that was not contributory to the deaths, however, increased from 1941 to 1952.

Table IV shows a marked increase in the frequency with which pelvic measurements were done, from 18% in 1941 to 46% in 1952. Nevertheless, measurements were not done or were incompletely done for more than half of the patients who died. Minimum pelvic mensuration as defined by the committee includes intertuberous diameter, diagonal conjugate, and palpation of sacrum and ischeal spines.

Serologic test for syphilis were done in only one-third (31.9%) of the cases for which deaths were reported in 1941; they were done in 56% in 1952 (Table V).

An increasing number of Rh determinations (63%) were done in 1952, especially early in pregnancy (Table VI). In 17% no Rh determination was made and in 19.5% it was obtained only when the patient entered the hospital for delivery.

Labor and delivery care showed marked improvement (Table VII). In 1941, fewer than 8% had adequate care; in 1952, slightly more than one-half (51%) had adequate care. Faulty care that contributed to patients' deaths decreased from 68% in 1941 to 9% in 1952. But in 1952, 40% had care that was faulty although not contributory to the deaths.

Postpartum care was considered adequate in only 5% in 1941

but increased to 55% in 1952. (Table VIII). Faulty care that contributed to the patient's deaths showed a marked reduction from 80% in 1941 to 31% in 1952. Nevertheless, almost one-third of the deaths had faulty care that contributed to the deaths.

Consultations were considered adequate in only 9% of the cases for which deaths were reported in 1941 (Table IX). In 1952, 52% were adequate. However, further consultation was indicated in some cases and in others consultation was obtained too late to be of value. Of the 22 cases in which no consultation was obtained in 1952, it was indicated, in the opinion of the committee, in only 8.

During each of the four years of the study more than half of the deliveries were operative (Table X). However, only three of the 19 operative deliveries or 16% were considered not indicated in 1952. This percentage compares well with the 44% not indicated in the years 1950 and 1951 combined.

The primary causes of death are listed on Table XI. It should be noted that 43% of the deaths were associated with pregnancy or the postpartum state and were due to non-obstetrical causes. Poliomyelitis accounted for as many deaths in the non-obstetrical group as hemorrhage, the leading cause of obstetrical deaths. Among the 33 obstetrical causes of deaths, hemorrhage was responsible for one-third (33.3%), toxemia for one-sixth (18%), followed by amniotic fluid embolism (6%) and air embolism (6%).

For the latest three years of the study (1950, 1951 and 1952), hemorrhage, the leading cause of death, was responsible for 28% of the obstetric deaths and caused by far the greatest number of preventable deaths, (44.6%) (Table XII). Hemorrhage accounts for more than three times as many preventable deaths as does the next leading cause, toxemia, which accounted for 14%.

Rupture of uterus and lacerations of cervix due to accouchement forcé and improper use of pituitary extract accounted for one-third of the deaths due to hemorrhage in the latest three years of the study. Postpartum uterine atony was responsible for one-fourth of the deaths due to hemorrhage. In practically all instances blood was available in adequate amounts. However, in all except three cases where death occurred, the blood was administered too late and/or in inadequate amounts.

It should be noted that anesthesia was responsible for twice as many preventable deaths as infection.

Analyses of maternal death certificates indicate that many certificates are incomplete and inaccurate, usually because of carelessness and occasionally not unintentional (Table XIII). There has been a marked increase in the number of correct certificates, from 36.6% in 1941 to 61% in the latest three years. But one-half of these are incomplete. More serious, however, is the fact

that in the Committee's opinion 28% of the certificates were incorrect as to cause of death.

Adequate autopsies were obtained in 38.8% of the deaths during the latest three years (Table XIV). While this percentage is a marked improvement over the 8% obtained in 1941 there is room for more improvement. Failure to obtain autopsies is due in the majority of cases to the physicians' neglect to request them. They were not requested in 49% in 1941 and in 38% during the latest three years.

A most significant achievement in 1952 was the reduction in the number of deaths considered preventable (Table XV). In 1941, 82 deaths or almost three-fourths (73.2%) were considered preventable; in 1952, only ten deaths or one-sixth (17.2%) were rated as preventable.

Surveys such as have been described have a desirable psychological effect on physicians and hospitals, as well as on the public. Physicians become more careful and more conservative in their treatment and are more likely to request earlier consultation.

It is important that the results of these studies should be used for the education and guidance of physicians at hospital staff meetings, at state and county medical society meetings, at refresher courses, for undergraduate teaching at the medical schools, and for publication in medical journals.

In spite of the great progress that has been achieved in making pregnancy and childhcod safe, there is evidence that there is still need for improvement. Maternal mortality surveys as well as neonatal mortality studies can play a significant role in such improvement. Such studies will lead to better obstetric and newborn techniques and practices. The ultimate result should be a further reduction in maternal and neonatal mortality.

TABLE I — OBSTETRIC AND NON-OBSTETRIC DEATHS
MINNESOTA SURVEYS OF 1941, 1950, 1951, and 1952.

	1941		1950		1951		1952	
	No.	Per cent	No.	Per cent	No.	Per cent	No.	Per cent
Obstetric deaths	93	83.0	46	67.6	39	68.4	33	56.9
Non-obstetric	19	17.0	22	32.4	18	31.6	25	43.1
Totals	112	100.0	68	100.0	57	100.0	58	100.0

TABLE II — LIVE BIRTHS, PER CENT IN HOSPITALS, MATERNAL DEATHS AND MORTALITY RATES, MINNESOTA SURVEYS FOR 1941, 1950, 1951, 1952

	Live Births	Per cent Births in Hospital	Maternal Study	Deaths Minn. V.S.	Maternal Mortality Rate Per 1000 Live Births: Gross Study**	Minn. V.S. ***	U.S.
1941-42 av.	55,293	76.0	112	93	2.03	1.6	2.8
1950	75,290	98.3	68	46	0.89	0.6	0.83*
1951	80,047	98.6	57	27	0.71	0.3	0.71*
1952	79,198	98.9	58	28	0.73	0.4	0.7*

* Unofficial.
** Including all deaths from whatever cause.
*** Excluding deaths considered by the Maternal Mortality Committee as non-obstetric.

TABLE III — PRENATAL CARE — MINNESOTA SURVEYS

	1941		1950		1951		1952	
	No.	Per cent	No.	Per cent	No.	Per cent	No.	Per cent
Adequate	2	1.9	15	24.2	6	13.6	10	21.3
Faulty, contributory	55	52.9	14	22.6	10	22.7	5	10.6
Faulty, not contributory	47	45.2	33	53.2	28	63.6	32	68.1
None	8		6		13		11	
Totals	112	100.0	68	100.0	57	99.9	58	100.0

TABLE IV — PELVIC MEASUREMENTS — MINNESOTA SURVEYS

	1941		1950		1951		1952	
	No.	Per cent of Registered Patients	No.	Per cent of Registered Patients	No.	Per cent of Registered Patients	No.	Per cent of Registered Patients
Unregistered	18		6		13		12	
Not measured or incompletely measured	77	81.9	40	64.5	32	72.7	25	54.3
Measured	17	18.1	22	35.5	12	27.3	21	45.7

TABLE V — SEROLOGIC TESTS FOR SYPHILIS — MINNESOTA SURVEYS

	1941		1950		1951		1952	
	No.	Per cent of Registered Patients	No.	Per cent of Registered Patients	No.	Per cent of Registered Patients	No.	Per cent of Registered Patients
Unregistered	18		6		13		12	
Not obtained	64	68.1	38	61.1	25	56.8	20	43.5
Obtained	30	31.9	24	38.9	19	43.2	26	56.5

TABLE VI — RH DETERMINATIONS — MINNESOTA SURVEYS

	1950		1951		1952	
	No.	Per cent of Registered Patients	No.	Per cent of Registered Patients	No.	Per cent of Registered Patients
Unregistered	6		13		12	
Not obtained (regist. pts.)	23	37.1	17	39.5	8	17.4
Obtained at hospital	11	17.7	10	23.3	9	19.5
Obtained early in pregnancy	28	45.2	16	37.2	29	63.0
No data			1			
Totals	68	100.0	57	100.0	58	99.9

TABLE VII — CARE IN LABOR AND DELIVERY — MINNESOTA SURVEYS

	1941		1950		1951		1952	
	No.	Per cent	No.	Per cent	No.	Per cent	No.	Per cent
Adequate	7	7.8	21	28.9	11	25.5	23	51.1
Faulty, contributory	61	67.8	26	48.1	23	53.5	4	8.9
Faulty, not contributory	22	24.4	7	13.0	9	21.0	18	40.0
None	22		14		14		13	
Totals	112	100.0	68	100.0	57	100.0	58	100.0

TABLE VIII — POSTPARTUM CARE — MINNESOTA SURVEYS

	1941		1950		1951		1952	
	No.	Per cent	No.	Per cent	No.	Per cent	No.	Per cent
Adequate	4	4.8	18	37.5	14	42.4	16	55.2
Faulty, contributory	67	79.8	27	56.3	15	45.5	9	31.0
Faulty, not contributory	13	15.5	3	6.2	4	12.1	4	13.8
None	28		20		24		29	
Totals	112	100.1	68	100.0	57	100.0	58	100.0

TABLE IX — CONSULTATIONS — MINNESOTA SURVEYS

	1941		1950		1951		1952	
	No.	Per cent	No.	Per cent	No.	Per cent	No.	Per cent
None	63	56.2	29	42.6	28	49.1	22	37.9
Not indicated			12		14		14	
Indicated			17		14		8	
Adeqnate	10	9.0	22	32.4	17	29.8	30	51.7
Inadequate	39	34.8	17	25.0	12	21.1	6	10.3
Totals	112	100.0	68	100.0	57	100.0	58	99.9

TABLE X — TYPE OF DELIVERY — MINNESOTA SURVEYS

	1941		1950		1951		1952	
	No.	Per cent of Delivered Patients	No.	Per cent of Delivered Patients	No.	Per cent of Delivered Patients	No.	Per cent of Delivered Patients
Undelivered	23		17		21		27**	
Spontaneous	39	43.8	24	46.2	16	44.4	13	40.6
Operative	50	56.2	28	53.8	20	55.6	19	59.4
Indicated			19		8		16	
Not indicated			9		12		3	
Totals	112	100.0	69*	100.0	57	100.0	59*	100.0

* One set of twins.
** Includes 7 delivered by post-mortem cesarean section.

TABLE XI — PRIMARY CAUSES OF MATERNAL DEATHS
MINNESOTA SURVEY, 1952

Cause of Death		No.	Per cent
Hemorrhage		11	19.0
Toxemia		6	10.3
Amniotic fluid embolism		2	3.5
Air embolism		2	3.5
Heart disease		2	3.5
Acute pulmonary edema		1	1.7
Septic abortion		1	1.7
Dehydration and electrolyte imbalance		1	1.7
Chorionepithelioma		1	1.7
Probable pulmonary embolus		1	1.7
Acute hepatitis		1	1.7
Unknown		4	7.0
Non-obstetrical complications		25	43.1
Bulbar poliomyelitis	11		
Trauma	4		
Intracranial hemorrhage	3		
Brain tumor	2		
Leukemia	1		
Suicide	1		
Obstruction trachea	1		
Acute glomerulonephritis	1		
Thrombocytopenic purpura	1		
Totals		58	100.1

TABLE XII — LEADING CAUSES OF "OBSTETRIC" DEATHS BY NUMBER AND PERCENTAGE, INCLUDING PREVENTABILITY — MINNESOTA SURVEYS, 1950, 1951 AND 1952

	No.	Per cent	Preventable	Per cent of Preventable Deaths
Hemorrhage	33	28.0	25	44.6
Toxemia	23	19.5	8	14.3
Infection	8	6.8	3	5.4
Anesthesia	7	5.9	6	10.7
Pulmonary embolism	7	5.9	0	0.0
Others	40	33.9	14	25.0
Total	118	100.0	56	100.0

TABLE XIII — ANALYSIS OF DEATH CERTIFICATES MINNESOTA SURVEYS

	1941		1950, 1951, 1952	
	No.	Per cent	No.	Per cent
Total maternal deaths	112	100.0	183	100.0
Correct & complete in all details	19	17.0	57	31.0
Correct in details reported	22	19.6	54	30.0
Incorrect	71	63.4	72	39.0
Incorrect as to cause of death			52	28.0

TABLE XIV — AUTOPSIES — MINNESOTA SURVEYS

	1941		1950, 1951, 1952	
	No.	Per cent	No.	Per cent
Obtained, adequate	9	8.0	71	38.8
Obtained, inadequate	27	24.1	6	3.3
Not requested	55	49.1	70	38.3
Permission refused	21	18.8	36	19.6
Totals	112	100.0	183	100.0

TABLE XV — PREVENTABILITY OF MATERNAL DEATHS IN MINNESOTA — MINNESOTA SURVEYS

	1941		1950		1951		1952	
	No.	Per cent	No.	Per cent	No.	Per cent	No.	Per cent
Preventable	82	73.2	22	32.4	24	42.1	10	17.3
Non preventable	27	24.1	45	66.1	32	56.1	47	81.0
Not determinable	3	2.7	1	1.5	1	1.8	1	1.7
Totals	112	100.0	68	100.0	57	100.0	58	100.0

STATISTICS ABOUT PHYSICIANS IN PUERTO RICO (1)
Jan. 1, 1955

O. COSTA MANDRY, M.D.*

CONTENTS

KEY TO SIMBOLS

Diplomate American Specialty Board.
° Took Board examinations, results unknown to us.
* Studying at present or has studied with P. R. Government scholarship.
✧ Studying at present or has studied with scholarship other than P. R. Government.
❖ In armed forces.

Table No. 1 — PHYSICIANS OF PUERTO RICO — Jan. 1, 1955

Distribution	Physicians			Population per Physician
	No.	% of total	% of subtotal	
All Physicians	1,290	100	—	1,736
In practice	1,021	79.1	100	2,192
General	597	46.2	58.4	3,750
Specialists	424	32.8	41.6	5,280
Employed in Health Department	312	24.2	30.5	7,176
Regular (and probatory)	190	—	—	—
Provisional	28	—	—	—
By clinic	47	—	—	—
Contract (Emergency Law)	47	—	—	—
Members P. R. Medical Ass.	672	52.1	—	—
Internes	80	6.2	—	—
Retired	6	0.46	—	—
Outside of Puerto Rico	32	2.5	—	—
Armed Forces	77	5.9	—	—
Studying for Specialty	74	5.7	—	—
Scholarship (P. R. Govt.)	9	—	—	—
Residents in P. R. Hosp.	40	3.1	—	—

Population P. R. - 7/1/54 - Increment Births - deaths, migration - 2,238,987

(1) Computed from figures in our possesion and from the requirements of the American Specialty Boards and our criterion as to what constitutes a specialist.

* Director, Office of Pathology and Medical Education, Dept. of Health, Santurce, P. R.

Table No. 2 — SPECIALISTS BY SPECIALTIES — Jan. 1955

Specialty	Physicians		American Boards		Studying	
	No.	%	Cert.	Pend.	U.S.	P.R.
All Specialties	424	100	118	17	34	40
I — MEDICAL SPECIALTIES	148	34.9	41	9	7	18
Internal Medicine	31	7.3	25	6	—	11
Hematology	3	0.7	(1-IM)			
Cardiology	12	2.8	2(6-IM)			
Gastro-enterology	7	1.6	(1-IM)	1		—
Allergy	3	0.7				
Tuberculosis	28	6.6				
Pediatrics	28	6.6	10	1	2	7
Dermatology	8	1.8	2			
Psychiatry-neurology	28	6.6	2	1	5	0
II — SURGICAL SPECIALTIES	145	34.1	42	3	11	9
General Surgery	66	15.6	15	3	2	8
Neuro-surgery	5	1.1	2	—	—	—
Orthopedics	11	2.6	6		1	—
Ophthalmology	20	4.7	7		4	—
Oto-rhino-laryngology	19	4.4	7		2	
Plastic surgery	2	0.4	(1-Surg.)			
Thoracic Surgery	4	0.9				
Urology	14	3.3	5		—	1
Proctology	1	0.2			1	
Cancerology	3	0.7			1	
III — OBSTETRICS-GYNECOLOGY	54	12.7	8	3	4	5
IV — PATHOLOGY	16	3.8	5(1-IM)	—	7	2
General Pathology	11	2.6	4	—	6	2
Clinical Pathology	5	1.1	1(1-IM)	—	1	1
V — RADIOLOGY	15	3.5	10	1	2	1
VI — ANESTHESIA	4	0.9	2	—	2	2
VII — PHYSICAL MEDICINE	4	0.9	1	—	1	—
VIII — PREVENTIVE MEDICINE	38	8.9	5	1	—	

Table No. 3 SPECIALISTS BY TOWN

Physicians	All	Metropolitan area*		Ponce		Aguadilla Arecibo Fajardo		Rest of island	
		No.	%	No.	%	No.	%	No.	%
Physicians in Practice	1,021	578	56.6	69	6.7	100	9.7	274	26.8
General Practitioners	597	245	41.0	36	6.0	79	13.2	237	39.6
Specialties	424	331	78.1	35	8.2	22	5.2	36	8.5
I—MEDICAL SPECIALTIES	148	124	29.2	10	2.3	6	1.4	8	1.8
Internal Medicine		29		1		1			
Hematology		2		1					
Cardiology		10		1		1			
Gastro-enterology		7							
Allergy		2		1				6	
Tuberculosis		18		3		1		2	
Pediatrics		20		3		3			
Dermatology		8						—	
Psychiatry-neurology		28		—		—			
II—SURGICAL SPECIALTIES	145	108	25.5	14	3.3	10	2.3	11	2.6
General Surgery		42		7		7		10	
Neuro-surgery		5							
Orthopedics		10		1					
Ophthalmology		17		2		1			
Oto-rhino-laryngology		15				1			
Plastic surgery		2						1	
Thoracic-surgery		3		1					
Urology		11		2		1			
Proctology		1							
Cancerology		2		1					
III—OBSTETRICS-GYNECOLOGY	54	42	9.9	4	0.9	2	0.5	6	1.4
IV—PATHOLOGY	16	14	3.3	1	0.23	—	—	1	0.23
General Pathology		9	—	1	—	—	—	1	—
Clinical Pathology		5	—	—	—	—	—	—	—
V—RADIOLOGY	15	12	2.8	2	0.5	1	0.23	—	—
VI—ANESTHESIA	4	3	0.7	1	0.23	—	—	—	—
VII—PHYSICAL MEDICINE	4	4	0.9	—	—	—	—	—	—
VIII—PREVENTIVE MEDICINE	38	24	5.7	2	0.5	3	0.7	9	

* San Juan, Río Piedras, Bayamón.

TABLE NO. 4 — SPECIALISTS BY NAME AND SPECIALTY

I — MEDICAL SPECIALTIES

INTERNAL MEDICINE

Juan E. Acevedo
Agustín M. Andino °
Carlos Bertrán °
Roberto Busó
José A. de Jesús #
F. Diez Rivas #
Lillian Ferrer
Hilda García de la Noceda
José R. González Flores
Jenaro Haddock #
Ulises López Sanabria
Víctor Malagón
Ernesto C. Martínez #*
Héctor Martínez Villafañe
R. Menéndez Corrada #
Eduardo Montilla
Manuel E. Paniagua °
Manuel Pavía Fernández
Eduardo R. Pons #
Juan A. Pons
M. Pujadas Díaz
Elí S. Ramírez #
Julio Rivera #
Zenón Rivera Biascoechea
Rafael Rodríguez Molina #
Calixto A. Romero ❖#
Andrés A. Salazar #
Ramón Suárez Benítez
A. Rivera Trujillo
Hiram Vázquez °
Raúl Vizcarrondo

Studying for Specialty

Raúl Acosta (Mun.)
J. Alvarez Calderón (San P.)
Ana L. Cordero (Mun.)
R. García Palmieri (Mun.)
Jorge Mayoral (San Patricio)
R. Méndez Bryan (Mun.)
Juan A. Nogueras (Mun.)
Héctor Rodríguez (Bayamón)
Roberto Rodríguez (Mun.)
Zoilo Sotomayor (Mun.)
Miguel A. Timothee (Bayamón)

HEMATOLOGY

Angel A. Cintrón
Enrique Pérez Santiago °* (IM)
Remy Rodríguez *# (IM)

CARDIOLOGY

José A. Amadeo
R. Arrillaga Torrens # (IM)
Rurico S. Díaz Rivera # (IM and Card.)
Roberto Francisco # (IM)
Ernesto J. Marchand # (IM)
Ezequiel Prieto
Dwight Santiago # (IM)
José A. Seín
Ramón M. Suárez # (IM and Card.)
Carlos E. Timothée
José M. Torres # (IM)
Federico Velázquez

GASTROENTEROLOGY

José Berio Suárez ❖*°
Bartolomé Borrás
F. Hernández Morales # (IM and GE)
José L. Robert
A. Rodríguez Olleros °
Ramón J. Sifre
Ramón Sifre Amadeo # (IM)

ALLERGY

Donald F. Gowe
Angel M. Marchand
J. Nieves Colón

TUBERCULOSIS

Carlos Armstrong
Juan Arruza
Luis J. Betances *
Fernando Buxeda
Ramón T. Colón
Pedro J. Durand
E. Fernández Cerra
Jorge Franceschi Juliá
José A. Franco (ret)
Jaime L. Fuster
David E. García
Luis García de Quevedo
Philip S. Gorlin
L. Jouanneau
J. J. de Lara García
Héctor A. Marrero
E. Martínez Rivera
J. M. Moscoso Rodríguez

Borinquen Mussenden
Fernando Padró *
José Luis Porrata
Alice Reinhardt Valcourt
J. Rodríguez Pastor
Hilarión Sánchez ❖
Manuel Santiago Santos
Leandro Santos
Jacobo Simonet
José Soto Ramos
Rafael Velázquez

PEDIATRICS

Raúl Acosta
Ricardo Alonso
Joseph Aponte
Lorenzo Balasquide
J. Basora Defilló #
Antonio R. Busquets #
Dolores M. Cashion #
M. Castro
Alberto Díaz Atiles
César Domínguez
Miguel A. Firpi, Jr. #
Lydia González de Montalvo
Benito Irizarry Bulls
E. Matta
Ceferino Méndez
Pedro Mendoza
Enrique A. Milán #
Eduardo Mirabal ❖
Eloísa Muñoz Dones
Juan J. Nogueras
Egidio S. Colón Rivera #
Antonio Ortiz #
Ydalia Ortiz #
Katheryn Rakesky Rivera
Salvador Riera López
Rafael Rivera
Mimosa M. de Rullán
Catalina Scarano
Jenaro Scarano #
José E. Sifontes * #
Víctor Skerrett
Dharma L. Vargas #

Studying for Specialty

Carmen Berio (Chicago)
R. Blasini (Mun.)
Margarita Cáceres (Mun.)
Arnaldo García (Mun.)
Plácido González Alvarez (Mun.)
Julieta Grana (Mun.)
Héctor Hidalgo (Mun.)
Juan Jiménez Mercado (Mun.)
Simón Piovanetti (Jefferson Med. Col.)

DERMATOLOGY

Alfredo L. Bou
Arturo L. Carrión #
José F. Correa
Honorato Estella
Víctor J. Montilla
Jesús M. Quiñones
Víctor M. Rivera #
Elí S. Rojas

PSYCHIATRY and NEUROLOGY

L. Alvarez Pou *
Víctor Arzuela
Rafael Arzuaga
Víctor Bernal y del Río *
Fernando M. Canino *
Rafael Cantón
Providencia Castro *
Mario C. Fernández
R. Fernández Marina *
Arturo Flores Gallardo *
José García de la Madrid *
Leopoldo García Mercado #
Juan Homedes
Ana Janer
Edwin Jiménez Pabón *
José D. Jiménez
Mario Juliá
Juan A. Mascort
Carlos L. Massanet
Teodoro Milán *
Luis J. Montalvo
Juan E. Morales #
Luis M. Morales #
José Olmedo ❖
Luis Ortega
María Robert de Ramírez
Juan Rosselló °
Ramón H. Señeriz
Luis del Toro *
R. Troyano de los Ríos

Studying for Specialty

Eduardo Maldonado Sierra (Phila.)
Rubén Nazario (Calif.)
M. Rodríguez Pérez * (U.S.)
Waldo J. Rodríguez * (U.S.)
A. Torres Aguiar * (U.S.)

II — SURGICAL SPECIALTIES

GENERAL SURGERY

Alberto Adams ᵒ❖
Ramón I. Almodóvar ❖
José J. Alonso
Fernando Asencio
Manuel A. Astor
A. L. Axtmayer #
Manuel Baralt
Guillermo H. Barbosa
Jenaro Barreras
Juan M. Bertrán Margarida #
Gumersindo Blanco #
E. Blás Ferraiuoli #
Salvador C. Busquets
Néstor de Cardona
Manuel G. Carrera
A. S. Casanova Díaz #
Marvin S. Cashion ᵒ
Rafael Coca Mir ❖
Arsenio Comas
Eugenio M. de Hostos #
Francisco R. de Jesús
Luis Díaz Bonnet ᵒ
José Forastieri
Manuel Garrido Carmona ❖
William R. Gelpí
Antonio R. Ginorio
Benigno T. González
J. González Giusti #
José F. González
Roberto Jiménez ᵒ
Carlos M. Juliá
Raúl Justiniano ❖
José S. Licha #
Anthony Lombardi
H. Luiggi
Iván Marqués ❖
Herbert Mayer #
R. Mejía Ruiz
C. B. Moore ᵒ
Justo Luis Muñoz
José Noya Benítez #
A. Oliveras Guerra
Arnaldo Palmer López
Luis A. Passalacqua #
Nelson Perea
Carlos A. Quilichini
Frank L. Raffucci #*
Arquelio Ramírez
A. Ramos Oller
Víctor Rincón
E. Rodríguez Pérez
Félix Rodríguez Forteza
J. M. Rodríguez Quiñones
Juan C. Rodríguez
Julio R. Rolenson
Joaquín Rovira Palés
Pedro J. Rullán
Luis F. Sala #
José Sárraga #
Américo Serra
José Sobrino
Roy J. Stokes
Pedro A. Suau #
Antonio Susoni
Dámaso Talavera
Mario J. Tomasini
Miguel A. Tulla
Luis A. Vallecillo #

Studying for Specialty

R. Arredondo Sánchez (Mun.)
José Bernal Rosa (Mun.)
Arturo Cadilla (Bay.)
A. Crespo (Bayamón)
Gilberto Fernández (San P.)
Juan L. López Morales (Mun.)
Roque Nido (Bayamón)
Pedro Otero (San P.)
Raúl R. Rivera (Milwaukee)
John F. Sanabria (Mun.)
Zaida Torres (U.S.)

NEUROLOGICAL SURGERY

J. Alvarez de Choudens
Ricardo Cordero
Luis R. Guzmán #*
Max Ramírez de Arellano
Nathan Rifkinson #*

ORTHOPEDIC SURGERY

Juan R. Cabrera *
J. Dávila López #
Manuel Espinosa Robledo
M. Guzmán Acosta *
José Iguina Reyes
Aníbal G. Lugo #
Antonio E. Molina #
Ian Murphy #
Peter E. Sabatelle #
León Sheplan #

Studying for Specialty

José Suárez Alvarez (Phila.)

OPHTHALMOLOGY

José L. Arbona
Rafael Bernabe
Héctor A. Bladuell
Roberto Buxeda #
H. F. Carrasquillo #
L. Cuello
Luis J. Fernández #
Ricardo F. Fernández #
J. A. Gallardo
Agustín R. Laugier
Andrés Montalvo #
Luis A. Morales #
Antonio Navas
Lydia Pérez Guardiola
Guillermo Picó #*
Nicolás Quiñones Jiménez, Jr.
Pedro Vargas Rosado
Jacinto Zaratt

Studying for Specialty

Carlos Margarida (Presb. Hosp., N.Y.)
J. Fiol Bigas (Phila.)
José Pietri (Milwaukee)
José Vidal (Phila.)

OTORHINOLARYNGOLOGY

Miguel Alonso #
Lorenzo Arsuaga
Adolfo Bernabe
Luis C. Clavell
Colby W. Dunscombe #
Jaime Font Casalduc
Juan H. Font #
Rafael Maldonado Quiñones
Miguel A. Mariani
Carlos E. Muñoz MacCormick #
A. Pérez Toledo
Eduardo R. Pérez
José Picó #
F. Quiñones Jiménez
N. Quiñones Jiménez
William Reichard #
Antonio Rullán #
Miguel A. Zapata

Studying for Specialty

J. Látimer (U.S.)
R. Magriñá Suárez
E. Vicéns (Phila.)

PLASTIC SURGERY

J. Benavent # (Surg.)
Angeles Díaz

THORACIC SURGERY

Gustavo Bergnes
Jaimes Costas Durieux
Jaime F. Pou
David Rodríguez Pérez

UROLOGY

Manuel F. Alsina Capó
Natalio Bayonet
Julio E. Colón
Rafael Colón
Pablo G. Curbelo #
José E. Dávila
José C. Ferrer
E. García Cabrera
José Gelabert
Antonio Guijarro ❖
Luis M. Isales #
M. Maeso
A. Mejía Casals #
Néstor H. Méndez #
B. Rodríguez Lucca ❖
Luis A. Sanjurjo #

Studying for Specialty

Roberto Fortuño (Mun.)

PROCTOLOGY

William P. Gelpi

Studying for Specialty

Miguel Valiente (U.S.)

CANCEROLOGY

José R. Casanova Díaz *
Hamlet Hazim
Ramón T. Llobet ✧

Studying for Specialty

Víctor Marcial (U.S.)

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Luis A. Amorós
E. Arandes Rexach
Roberto L. Axtmayer❖
Luis Bartolomei
Fernando A. Batlle
José S. Belaval (retired)
Elwood M. Bond
José Díaz Carazo°
Modesto Carrasquillo
José B. Caso
Alberto Castañer
Angel R. Cestero #
Miguel A. Cintrón
María E. Cora
Jeramfel Cordero
Miguel S. Dalmau
M. Fernández Fuster #
Leopoldo Figueroa
Andrés Franceschi
A. García Castillo
A. García Soltero
J. García Bird #
J. García García
José García Blanco
Rafael A. Gil #
Josefina Guarch°
Jorge Hereter #
Manuel A. Iguina #
J. B. Kodesh #
Blanca Lluberas
Antonio Medina
L. Von Misky
Juan Mimoso
Randolph J. McConnie #
María A. Parés
Iván Pelegrina
J. Salvador Plá
M. Rodríguez Ema
Carmen Romero Gelpi
José A. Roure
Elvira Rodríguez
E. Rodríguez Pacheco
Roberto Serra
E. Schroder
M. Soto Rivera
Jenaro Suárez
Luis Torres Oliver
José H. Vázquez
José Luis Vilá
Rafael A. Vilar
Edgardo Yordán

Studying for Specialty

M. T. Berio (U.S.)
S. Colón Lugo (Mun.)
Isabel V. Estrada (U.S.)
M. Fernández Durán (Mun.)
J. García Esteves (Mun.)
Hiram Gómez (U.S.)
Alejandro Infanzón (U.S.)
Paul Mari (Mun.)
Radamés Orlandi (Mun.)

IV — PATHOLOGY

Clinical Pathology

O. Costa Mandry # (CP and IM)
F. J. Mejías
P. Morales Otero
R. Ruiz Nazario
Luis A. Salivia

GENERAL PATHOLOGY

Víctor Arean
Donald F. Babb #
Donald R. Jutzy
Enrique Koppisch #
Francisco Litchtenberg #
Raúl A. Marcial Rojas *#
Jorge B. Morales *❖
Esteban Moreno ❖*
Bolívar Patiño *
Gerardo Polanco *
Félix M. Reyes #
I. Rivera Lugo
J. E. Taveras

Studying for Specialty

Gonzalo Aponte (Phila.)
Walter Cervoni (Phila. Clin. Path.) *
Manuel de Jesús (Wash. DC)*
Eric Everts (Jefferson Med. Coll., Phila.)*❖
Lorenzo Galindo (Wash.)
Luis E. González Saldaña (Mun.)
R. Ramírez Weiser (Temple Univ.)*
Edwin Rivera (Mt. Sinai Hosp. - N.Y.)°*
Bolívar Patiño (Bayamón) Clin. Path.*)
Gustavo Ramírez de Arellano (Sch. Med. - P.R.)

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Rafael A. Blanes
Rafael B. Díaz Bonet*#
Lazlo Ehrlich #
Carlos Guzmán Acosta #*
M. Guzmán Rodríguez
Carlos Jiménez #
José Landrón º
Juan R. Marchand #*
Guillermo Marqués
Pablo Luis Morales #
R. Pérez Ribié #❖
Luis Ramos González ❖
Pedro Ramos Casellas #
Jesús Rivera Otero #*
Guillermo Ruiz Cestero
Héctor Vallés #
Luis A. Yordán

Studying for Specialty

Francisco Orobitz (U.S.)
Oscar Ruiz (U.S)
Tomás Jiménez Becerra (Mun.)

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E. Colón Yordán #
Miguel Figueroa ❖
Frederick J. González #
Paul Leni

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Francisco Bajandas (Mun.)
Carmen Castro Suárez (U.S.)
Celeste Castro (U.S.)
Ariel Méndez (Mun.)

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Herman J. Flax *#
Gabriel Rigau
Harold Storms

Studying for Specialty

Aureo Calderón (U.S.)

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Renato Royo *
J. H. Saavedra *
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Victoria Smith
Rafael Timothée *
Luis Torres Aguiar
Blanca H. Trelles de Vázquez
Rafael S. Vázquez

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Ramón Vázquez Pastrana (U.S.)

TABLE NO. 5 — INTERNES

SAN JUAN MUNICIPAL HOSPITAL

1. Aguayo Disdier, Rafael
2. Bauzá Hernández, Antonio
3. Berrocal, Enrique
4. Blasini Rivera, Marino
5. Defendini, Efraín A.
6. Medina, José T.
7. Menay Jusino, Abelardo
8. Pérez Arzola, Miguel
9. Piñot Nin, Ramón A.
10. Quiñones, Víctor Manuel
11. Ramírez Silva, Jr., Amador
12. Rivera Rodríguez, Freya
13. Rodríguez Negrón, Luis A.
14. Tur Rodríguez, Luis A.

ARECIBO DISTRICT HOSPITAL

1. Abréu Román, Antonio
2. Masari Sánchez, Ferdinand
3. Miranda, Ada
4. Parrilla, Eduardo
5. Rivera Rodríguez, Rodolfo
6. Soler Zapata, Juan H.
7. Somohano Mosquera, Angel M.
8. Vega Soto, José Rafael
9. Vicenty, Wassilly

BAYAMON DISTRICT HOSPITAL

1. Cerra Quiñones, Domingo
2. Cortés, Félix M.
3. Fores, Sally E.
4. Garriga Prida, Rafael
5. González Alcover, Rafael
6. Haddock Suárez, Lilliam
7. Machuca, Fernando
8. Picó, José F.
9. Rosa Silva, Héctor A.
10. Serra, Rafael E.

FAJARDO DISTRICT HOSPITAL

1. Anduze Menéndez, Julio
2. Camuñas, María I.
3. Nieves Calcaño, Gilberto
4. Piñeiro, Ramón
5. Ramos Yordán, Federico
6. Reyes Matos, Félix
7. Silva Monje, Luis D.
8. Torres, Luis A.

PRESBYTERIAN HOSP. (S.J.)

1. Bosch, José
2. Borrás, Pedro A.
3. Bursian, Tirso
4. Rivera, José H.
5. Rubio, Luis A.

ASILO DE DAMAS HOSP. (Ponce)

1. Bonín-Font, Pedro J.
2. Canabal López, Manuel
3. Clavell, Iván
4. López Vicario, Luis
5. Pérez Roig, Manuel
6. Vázquez, Joseph Gilberto

DR. SUSONI HOSPITAL (Arecibo)

1. Collazo Camuñas, R. E.
2. López Saavedra, Wilfredo

CLINICA PILA (Ponce)

1. Becerra, J.
2. Llavona, Jr.
3. Soler, J.
4. Visot, A.

AUXILIO MUTUO HOSPITAL (R. P.)

1. Collazo, J.
2. González, E. Raúl

PAVIA HOSPITAL (San Juan)

1. Sánchez Santiago, José E.

MUNCIPAL HOSPITAL (Rio Piedras)

1. Carlos, Ramón
2. Febles, Erasto
3. García Jiménez, Miguel
4. Guerrero, Rafael
5. Hart Messon, Humberto
6. Lebrón, Domingo
7. Martínez Cancel, Zenobio
8. Nieves, Pedro L.
9. Rojas Davis, Eli
10. Soto Respeto, Pedro
11. Vargas, Nicolás

DOCTOR'S HOSPITAL (Santurce)

1. Fernández, Ramón
2. Latoni, José
3. Pablos, G.

AGUADILLA DISTRICT HOSPITAL

1. Feliberty, Manuel
2. Montes Esteves, S.
3. Pérez Montalvo, J.
4. Piñero, B.

TABLE NO. 6 — RESIDENTS

BAYAMON DISTRICT HOSPITAL

1. Dr. Arturo Cadilla
2. Dr. A. Crespo
3. Dr. Adolfo Dacosta
4. Dr. Francisco J. Echegaray
5. Dr. Manuel Guzmán
6. Dr. Roque Nido Stella
7. Dr. Angel M. Otero
8. Dr. Miguel A. Timothée

FAJARDO DISTRICT HOSPITAL

1. Dra. Lydia D. Nieves Préstamo de Reyes
2. Dr. Ismael Ruiz
3. Dr. Juan José Vilella

ARECIBO DISTRICT HOSPITAL

1. Dr. Gabriel Magraner Folch
2. Dr. Pedro Mora Boneta
3. Dr. Angel Manuel Somohano

AGUADILLA DISTRICT HOSPITAL

1. Dr. Juan R. Vilaró

SAN PATRICIO HOSPITAL

1. Dr. J. Alvarez Calderón
2. Dr. Pedro Otero
3. Dr. L. Rodríguez Santos

MUNICIPAL HOSPITAL OF SAN JUAN

1. Dr. Raúl Acosta
2. Dr. R. Arredondo
3. Dr. Francisco Bajandas
4. Dr. José Bernal Rosa
5. Dr. Rafael Blasini Santiago
6. Dra. Margarita Cáceres
7. Dr. S. Colón Lugo
8. Dra. Ana L. Cordero
9. Dr. M. Fernández Durán
10. Dr. Roberto Fortuño
11. Dr. Arnaldo J. García
12. Dr. J. García Esteves
13. Dr. M. García Palmieri
14. Dra. Julieta Grana
15. Dr. Luis E. González Saldaña
16. Dr. H. Hidalgo
17. Dr. Tomás Jiménez Becerra
18. Dr. Juan Jiménez Mercado
19. Dr. J. Lionel López
20. Dr. Paul Mari
21. Dr. R. Méndez Bryan
22. Dr. Ariel Méndez
23. Dr. Juan A. Nogueras
24. Dr. R. Orlandi
25. Dr. Roberto C. Rodríguez
26. Dr. Alfonso Rozo
27. Dr. J. F. Sanabria
28. Dr. Zoilo Sotomayor

MUNICIPAL HOSPITAL (Rio Piedras)

1. Dr. Darío Delnero Riera
2. Dra. Rosa E. Fiol
3. Dr. A. Ruiz
4. Dr. Rubén Román
5. Dra. Amalia Sabater
6. Dr. L. Valdés

AUXILIO MUTUO HOSPITAL (Rio Piedras)

1. Dr. Antonio Berio
2. Dr. Gilberto Fernández

PRESBYTERIAN HOSPITAL (Santurce)

1. Dra. Rosa Serrano Solís

TABLE NO. 7 — RATIO INTERNES AND RESIDENTS TO HOSPITAL BEDS

	No. of beds	Ratio beds to		Actual Number*	
		Internes	Residents	Internes	Residents
Bayamón district †	250	25		10	
Medicine *	60	—	30		2
Obstetrics	60	—	30		2
Surgery *	70	—	23		3
Pediatrics *	60	—			—
Clinical Pathology *		—			1
Arecibo district †	250	25		8	3
Fajardo district †	250	25		8	3
Aguadilla district	250	60		4	1
Presbyterian †	150	30		5	1
Municipal - San Juan †	340	21		15	
Medicine *	90	13			7
Surgery *	90	22			4
Pediatrics *	80	12			7
Ob.-Gyn. *	80	20			4
Radiology *	—	—			1
Anesthesia	—	—			2
Pathology *	—	—			1
Damas - Ponce	150	25	—	6	—
Pila - Ponce †	150	40	—	4	—
Río Piedras - Municipal	150	15	25	10	6
San Patricio	200			—	
Medicine *	100	—	100		1
Surgery *	100	—	50		2
Auxilio Mutuo	200	100	100	2	2
Clínica Susoni	150	75	—	2	—
Hospital Pavía	150	150	—	1	
Doctors Hospital	150	50	—	3	

Approved AMA - CMEH - Internes † Residents *

TABLE NO. 8 — PHYSICIANS IN ARMED FORCES

Alemañy, Carlos E.
Almodóvar, R. (Surg.)
Arroyo, Pedro (USPHS)
Armstrong Mayoral, Raúl
Avilés, Juan
Axtmayer, R. (Ob.-Gyn.)
Aybar, José A.
Benítez, Rafael (Path.)
Berio Suárez, José (GE)
Berrocal, Carlos S.
Clark, S.
Colón Morales, Miguel A.
Coca Mir, Rafael (Surg.-USPHS)
Cook, Thomas A.
Dávila Polanco, José B.
De Juan, Abel Jr.
Díaz Montañez, Angel
Figueroa, M. (Anesth.)
Fox, Winslow G.
García, Iván H.
Garrido Carmona, Manuel (Surg.)
Garriga, José
Gelys, Alberto
González Alvarez, Plácido
González, Bernardino
González Jiménez, Enrique
Guardiola, Pablo
Guijarro, Antonio (Urol.)
Higgins, Peter O.
Justiniano, R. (Surg.)
Lacot Salgado, A. (Ob.-Gyn.)
Márquez Rivera, Iván (Surg.)
Martínez, Jorge L.
Mercado Jr., Raúl (Radiol.)
Matos, Angel L.
McDonald, Sylvester
Milán, Teodoro (Psych.)
Mirabal, E. (Ped.)
Montalvo, Eladio
Moreno, Eusebio (Path.)
Moreno, Fernando
Muñoz Candelario, A. L.
Murray, Geo. E. (Urol.)
Olmedo, José C. (Psych.)
Ortiz, Julio A.
Pérez Ribié, E. (Radio.)
Pouymirou, Frank
Quiñones, Rafael E.
Ramos González, Luis (Radiol.)
Romero, Calixto (Int. Med.)
Ramos Isern, F.
Rivera Ayala, G.
Rivera, Gerant
Rivera Pratt, J. E.
Rodríguez Lucca, Benigno (Urol.)
Rodríguez, Roberto C.
Rodríguez Rosado, Samuel
Romero, Angel
Rosa Febles, César
Roselló, Salvador
Rullán, José A.
Sais, Carlos
Salazar Rodríguez, José
Sabater, Homero
Salcedo, José R.
Salichs, Orlando
Sánchez, Hilarion (Tbc)
Sánchez Longo, Luis (Neurol.)
Sánchez Quiñones, A.
Simons, Julio
Sosa, José A.
Vázquez, Iván A.
Vera Sánchez, Ramón W.
Vilaró, Juan
Zamora, Pablo Luis

TABLE NO. 9 — FOREIGN PHYSICIANS WITH SPECIAL LICENCES WORKING IN HEALTH DEPARTMENT

1. Achecar, Felipe
2. Alonso Cervantes, Ernesto
3. Alonso Martínez, Ricardo
4. Arzuela Nido, Víctor M.
5. Calcagno Romano, Mario
6. Cantón, Rafael
7. Carle, Agenor Simeón
8. Carrera Giral, Jorge
9. Cheij Kourie, Abraham
10. Delgado Matallang, Gustavo
11. Díaz Martínez, Rafael
12. Duverge, Héctor A.
13. García, José R.
14. García Carrasco, Félix
15. García Galarza, José A.
16. García de Quevedo, Orlando
17. García Madrid, José M.
18. Gil, Julio Andrés
19. González Mañón, Jacinto
20. Gutiérrez Aliaga, Manuel
21. Hammerschmidt, César
22. Jouanneau Quincocés, Luciano
23. Lizardo Vidal, Francis
24. Llompart García, Juan
25. Macossay Negrín, Carlos
26. Mainardi, Luis E.
27. Martínez Martínez, Isidro
28. Mendoza Mendoza, Pedro
29. Molina Pérez, Rafael A.
30. Monserrat, Antonio
31. Montalvo Cordero, José H.
32. Moscoso Cordero, Juan M.
33. Najul Bez, Elías
34. Navarro, Francisco
35. Patiño Arca, Bolívar
36. Richards, Lawrence Leo
37. Sánchez Quintana, Juan
38. Solé Massana, Jorge
39. Santos Pérez, Eduardo
40. Tejada, Cornelio
41. Thomasa Sánchez, J. M.
42. Valdés, Ramón
43. Vázquez San Martin, José #
44. Villalón Ojeda, Conrado
45. Yapor Elías, Alfredo

TABLE NO. 10 — PHYSICIANS OUTSIDE OF PUERTO RICO

Norberto Acevedo	Medicine
A. Acosta Velarde	Tb
Guillermo Acosta	General Medicine
Roberto Aguayo	Pediatrics
María C. Barreras	Obs. and Gyn.
Walter Bond	Pathology
Tomás Cajigas	Pathology
Guillermo M. Carreras	Pathology
Jorge V. Crespo	General Medicine
Carlos Dalmau	Psychiatry
Carlos M. de Castro	General Medicine
Marina Díaz de Lee	Pediatrics
Felipa Díaz Santini	General Medicine
Carlos Domínguez	Surgery
R. Fernández Marchante	Pediatrics
A. Fernós Isern	Cardiology
Luis González Ramírez	Prev. Medicine
Rafael Hernández	Psychiatry
Manuel Janer	General Medicine
Daniel Landrón	General Medicine
Edward O'Neill	General Medicine
Sergio S. Peña	General Medicine
Agustín Pietri	Surgery
Carlos Pons	Pathology
Sarita Pons	Pathology
Edwin Rodríguez	Orthopedics
Eugenio Rodríguez	Pathology
Juan J. Santos	General Medicine
José Sugrañes	Radiology
Manuel L. Valdés	General Medicine
Emilio Vadi	Surgery
José G. Valderas	Obs. and Gyn.



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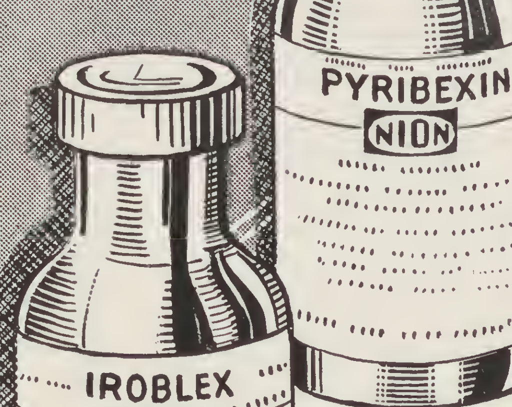


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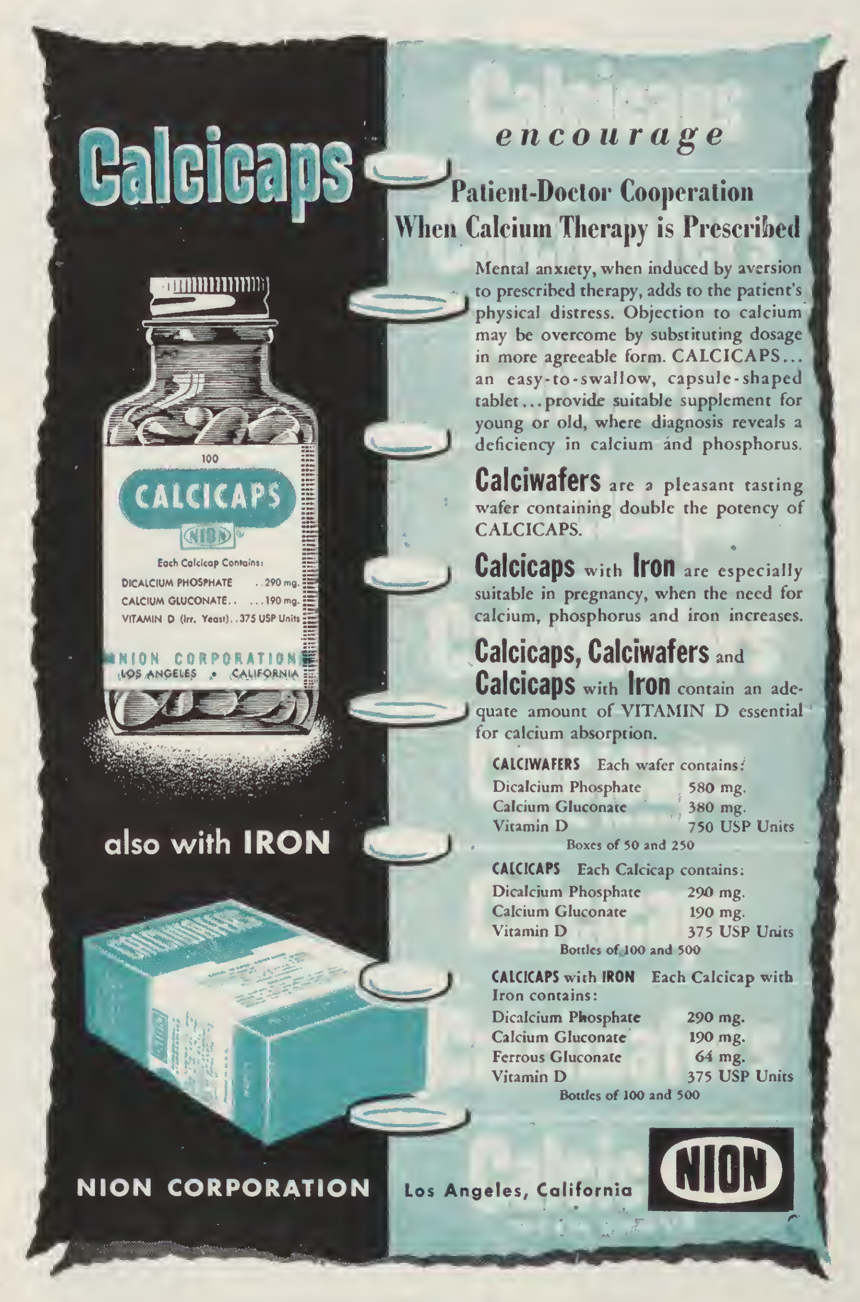
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†Steinberg, C. L., and Roodenburg, A. L.: J.A.M.A. 149:1458, 1952.
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TRANSANAL ILEOSTOMY*

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The diversion of the fecal stream with formation of an artificial anus is a problem frequently met with by the surgeon. When the terminal bowel is a portion of the colon, the solution is fairly simple, usually well accepted by the patient, and easily handled by the average surgeon. But when the terminal bowel is the ileum, then the problem becomes extremely serious. An ileostomy is not easily accepted nor tolerated by the patient, is frequently followed by many complications, and is the source of many worries for the surgeon. The comfort of the patient is interfered with and, at least, some of his outdoor activities are necessarily limited.

The formation of the usual abdominal ileostomy requires the constant use of a bag to collect the unrestrained, uncontrollable evacuations. To this, the average patient gets adapted reasonably well, and, in fact, he or she is mentally prepared to accept it willingly before actually undergoing the surgical procedure, provided his or her disease has been the source of great physical sufferings, as is usually the case with patients having complicated chronic ulcerative colitis. To other patients, though, whose preoperative condition has not been as precarious, the thought of an abdominal ileostomy with its associated ileostomy bag and discomforts, is not so attractive. And it would be much less attractive if the patient were in a position to know of the relative frequency of complications associated with such surgical procedure.

Among the more frequent and important complications of an abdominal ileostomy, we have the following:[5]

1) Prolapse of the stump; 2) retraction of the stump; 3) gangrene of the stump; 4) strictures with varying degrees of ob-

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struction; 5) perforations with resulting peritonitis, abscess, and/or fistula; 6) hemorrhage; 7) acute electrolyte imbalance; 8) nutritional disturbances; 9) skin irritation, excoriations, and ulcerations; and, 10) abdominal cramps.

With a better knowledge of intestinal physiology and nutritional requirements, improvements in anesthesia and in pre-and post-operative care, as well as in surgical technique, these complications have been gradually conquered to a satisfactory degree.

Prolapse of the stump is avoided quite well by fixation of the mesoileum to the parietal peritoneum,[1] care being taken not to encroach upon the circulation of the stump. If the circulation is interfered with, either by the fixation sutures, or during the mobilization of the portion of ileum to be exteriorized, then, prompt failure of the surgical procedure is inevitable because gangrene of the stump will follow, and will render another operation unavoidable.

Retraction is prevented by having a long viable stump projecting above the skin level. The stump, to start with, should be at least 2 or 3 inches long. It is better to have it too long than too short. If too long, it can always be cut down to the desired length. But a short stump is very annoying, and may precipitate the failure of the surgical procedure. The adaptation of a bag would be inefficient, and there would be leakage about it, with consequent skin excoriation and ulceration. If the retraction is too below the skin level, then peritonitis, wall abscesses or fistulae will follow.

To correct retraction and its associated complications, there is only one way: the formation of a new ileostomy, either at the same size as the previous one, or at a new site, depending on the local skin conditions.

Strictures may be due to various causes, most of which are the result of faulty surgical technique. The peritoneum, fascia or skin may have been closed too tight about the ileum; or the line of division of the ileum may have been through diseased bowel which eventually becomes too narrow for proper function; or the intestine may have been exteriorized with a severe twist. But, even avoiding these pitfalls, a stricture may be formed, and its control will require a careful follow up of the case, with dilatations of the stoma as frequently as may be necessary.

In cases of acute obstruction, the introduction of a small catheter into the ileum followed by careful irrigations, may be quite helpful. If these conservative measures fail, a revision of the ileostomy will be unavoidable.

Perforations are likely to be due to one of several causes: 1) residual disease in the ileum; 2) a suture which has been taken

through the bowel wall itself; 3) pressure from an indwelling rectal tube or catheter; or 4) interference with circulation of the bowel, with resulting focal gangrene. Increased intraluminal pressure from distal obstruction will hasten the perforation.

The perforation may be within the peritoneal cavity, with consequent peritonitis; or, within the abdominal wall, with consequent abdominal wall abscess and, eventually, the formation of a fistula; or, in the portion of the ileum projecting out from the skin. In the latter event, an "exteriorized" fistula will form, which will require revision of the ileostomy if the distance from the skin to the fistula is too short for a satisfactory application of the ileostomy bag after amputation of the stump at the level of the fistula. Suturing that type of perforation is practically always a waste of time because it usually recurs.

Of course, urgent surgical intervention is required for perforations within the peritoneal cavity or the abdominal wall.

Hemorrhage from the ileum may be due to residual disease in the retained intestine, to trauma from an indwelling tube, or to nutritional deficiency (mainly vitamin deficiency). A diseased intestinal mucosa will not tolerate the constant evacuations which are associated with an ileostomy, at least during the early postoperative period.

To prevent bleeding, all the diseased ileum should be removed (the line of resection of the ileum should be well within normal bowel); the indwelling tube should be handled with gentleness, its position changed frequently, and it should not be left in place any longer than necessary; and, nutritional deficiencies should be promptly corrected.

Once massive bleeding occurs, and fails to respond to conservative measures, then surgical intervention is required, with resection of more ileum.

Acute electrolyte and water imbalance is a very serious complication which occurs mainly during the immediate post-operative period. The patient may die so quickly from it, that the surgeon cannot afford to waste any time at all while preventing and correcting this imbalance. Accurate measure of the ileostomy and the urinary outputs is essential. The fixation of a narrow rectal tube or catheter into the ileal stump during the first several postoperative days, is not only a great help in keeping tract of the ileal output, but also it seems to, in some way, reduce the evacuation activity of the ileum, and, thus, reduce the amount of electrolytes and water lost. Besides, it helps to keep the skin and wound clean and free from irritation during the first days.

The general appearance of the patient, the relative moisture or dryness of the skin and mucous membranes, the turgidity of

the subcutaneous tissues, the ability and desire of the patient to move about and be active, the direction and abruptness of the weight curve, and the use of laboratory tests (sodium, potassium, chlorides, CO_2 combining power, red blood cells, and hematocrit determinations) help the physician to evaluate the true electrolytic state of the patient, and to determine which electrolytes, and how much of them, to give orally and parenterally. One must always remember that the sodium and potassium loss may be quite considerable.

Even with an apparently good electrolyte and water balance, the ileostomy patient may be a serious nutritional problem. He may fail to gain weight because of an inadequate absorption of food elements. The predigested protein products (protein hydrolysates) are a great help, given preferably by mouth, but, if necessary, by vein. A balanced, high caloric, high protein, low residue diet, well tolerated by the patient may be quiet difficult to arrive at.

The skin irritation and excoriation has been one of the worse problems to solve in the management of ileostomy patients. It is easier to prevent than to treat. Once it occurs, it may be quite discouraging to both patient and surgeon. Fortunately, the improvements in the preparation and application of ileostomy bags, and the easy availability of pharmaceutical preparations to protect the skin, have solved this problem to a great extent.

During the first 4 to 8 post-operative days, the presence of a well functioning ileostomy tube (inserted too well within the intraperitoneal segment of bowel) keeps the skin dry and clean. After that, the skin protection will depend on a well fitted bag, and on protective substances applied to the skin. Among the bags, there are provisional bags to be worn during the early post-operative period (such as the travellor bag), and permanent bags (Koenig-Rutzen; Torbot; Gerson-Jonas). Among the protective skin preparations we may mention 10% aluminum paste, kaolin-cod liver oil, zinc oxide ointment, zinc stearate powder, and covicone cream. The use of compound tincture of benzoin as a base layer before the application of any of the above preparations is quite helpful and should not be forgotten.

Abdominal cramps due to active peristalsis (not associated with a definite obstruction) may be quite bothersome during the first few weeks after the operation, but they usually subside regardless of the therapy used. Tincture of belladonna, paregoric, and anticholinergic drugs such as banthine, pro-banthine, wyovin and antrenyl, are helpful to a certain extent, but, not infrequently, demerol, or similar potent narcotics, may be required, though they should be used judiciously.

From all this discussion, we can truthfully conclude that ileostomy is an undesirable surgical procedure which should be resorted to only when really indicated, even though with the modern advances in medicine, a permanent ileostomy is not as troublesome as it used to be. Fortunately, the indications for a permanent ileostomy are few. Among these, the outstanding ones are: 1) Chronic ulcerative colitis, and 2) diffuse polyposis of the entire colon. Other conditions which may require ileostomy are: 1) multiple carcinomas of the colon and rectum, and 2) megacolon involving the whole colon.

We accept the first three of these four conditions as "bona-fide" indications for ileostomy, but, in the fourth, the megacolon, we would be very reluctant to recommend that type of operation. We believe that this condition can be corrected without resorting to an ileostomy.

We are not stating, either, that all cases of chronic ulcerative colitis, diffuse polyposis, and multiple carcinomas of the colon and rectum require permanent ileostomy. We just want to say that, in some of these cases, ileostomy is definitely indicated, but we do not want to enter into a discussion of the individual indications for permanent ileostomy in each one of these diseases. Suffice it to say that we feel that, when a total colectomy is indicated in any of these conditions, and the rectum can be preserved without endangering the life or well-being of the patient, an ileorectostomy is much preferred to any type of permanent ileostomy. But when the rectum is involved in the disease process to such an extent that it would be unwise to leave it in, then a permanent ileostomy should be mandatory.

Now, we have stated that a permanent ileostomy is undesirable, and, therefore, if we could accomplish a total removal of the colon and rectum and, yet, leave the patient free from the nuisance of an abdominal ileostomy, then something would have been accomplished.

To that effect, several attempts have been made to anastomose the ileum to the anal canal, thus forming an ileoproctostomy, which, nevertheless, could be regarded as an ileostomy. Nessin (1933), and Wangensteen and Toon (1948), reported on such a procedure, but it has been the work of Ravitch and Sabiston[6] who has given impetu to this form of treatment.

Devine and Webb,[4] and Best,[1, 2] have reported on modifications of the Ravitch procedure with variable degrees of success.

We have performed three cases since 1952, using a somewhat modified Ravitch procedure which we like to call "transanal sleeve ileostomy".[3] We start with the perineal (transanal) part of the operation, liberating several inches of rectal mucosa. Then we

change gowns, gloves, drapes, etc., and, proceed with the abdominal part through a long left paramedian incision.

After liberating the colon and part of the terminal ileum, with division of the latter between clamps, and after elevating the rectosigmoid and rectum from above, then we do a circumcision of the lower rectosigmoid down to the mucosal layer, separating the mucosa from the muscularis of the lower rectosigmoid and rectum to meet the line of mucosal dissection previously prepared during the perineal part of the operation, thus completely detaching the specimen to be removed.

It should be mentioned now, that it is preferable to start the circumcision of the rectosigmoid either laterally or posteriorly. The anterior wall is much thinner than most surgeons realize, and, therefore, to avoid cutting through the mucosa, it is better to start working on the thicker parts of that segment of bowel. The mucosal dissection can then be accomplished with more ease.

It should also be stated that in cases of chronic ulcerative colitis, no such thing as separation of the mucosa can be done because the mucosa is at least partially, destroyed by the disease, and, furthermore, the inflammatory changes render the dissection impossible. We feel that in those cases, part of the muscular wall should be removed to insure the removal of the diseased mucosa.

After the rectal "shell" (or "muscle tube") has been prepared, the divided end of the ileum, with a rectal tube tied into it, is inserted into the muscular "shell" and pushed out through the anus, where an assistant grabs it and pulls on it gently. At the same time, traction is exerted on the cut end of the rectal cuff so as to prevent its being intussuscepted, and, thus, shortened.

The assistant pulls gently on the ileal end until, at least 1½ to 2 inches of ileum project from the anus, which, of course, means that the terminal mesoileum has to be well liberated to allow such mobilization of ileum without endangering its circulation.

During this manipulation, the surgeon is watching the ileum and sees how it falls in the pelvis, and is able to avoid any undue traction or rotation. Then, the ileum is sutured to the rectal cuff, using, preferably, more than one row of sutures, and being careful to avoid penetration of the ileal mucosa with the sutures, as well as any damage to the intrinsic circulation of the ileum. And, by having made sure that the "rectal cuff" has not been wrinkled or intussuscepted prior to the suturing, we aim to accomplish one thing, namely, the prevention of undue retraction of the "pulled-through" ileum. This retraction is further prevented by suturing the prolapsed portion of ileum to the anal skin with a few sutures, after the abdominal part of the operation has been completed.

In one of our three cases we added something else to the procedure. The seromuscular layer of the segment of ileum which was inserted into the rectal "shell" was incised down to the mucosa, both longitudinally at the antimesenteric border, and circularly at different levels (but avoiding the blood vessels). This we did with the idea in mind that, by cutting down on the peristaltic activity of that segment of ileum, we could cut down on the number of bowel movements during the immediate post-operative period, and accelerate the adaptation of that segment of bowel to its new function as a substitute for the rectum. And, may be it is a coincidence, but the patient on whom we did that (E.A.C.), has, at the present time, the best functional results of the three, even though she developed some "intrarectal" infection during her early convalescence. Now she has 2 or 3 bowel movements during the daytime, and may be one at night. Continence, as in all three of them, is perfect.

In our short series of cases, we have encountered the following complications: 1. crampy abdominal pains. These were fairly severe in the first case operated on, practically totally absent in the second, and moderately severe in the last case. They lasted, off and on, for about two weeks. They did not respond too well to the usual antiperistaltic depressants (atropine, banthine, wyovin, etc.), and, when severe, required the use of narcotics (paregoric, demerol).

2. **Frequent bowel movements.** This is to be expected in all ileostomy cases, abdominal or otherwise, and may range from 10 to over 20 in 24 hours, during the immediate post-operative period. In our cases, the number of movements were considerably reduced in from a few days to a few weeks post-operatively, and they have continued to show a decided tendency to a progressive though slow reduction as time goes by. It is expected that, eventually, they will be reduced to from three to five or six movements per day.

In our second case, the girl of 13 on whom we tried the longitudinal and multiple circular incisions of the seromuscular coats of the ileum, we had a different course of events as far as the frequency of bowel movements is concerned. She had only 3 or 4 movements daily for the first 10 to 14 days postoperatively.... and had normal sphincteric control even that early. Unfortunately, the mobilized terminal ileum, which had been slightly too short, just barely reaching the outer border of the anus, retracted a little above the desired level, causing a separation between the intrarectal ileum and the posterior rectal wall. Consequently, a dead space resulted, followed by infection, which nevertheless, did not extend beyond the intrarectal space itself, but caused a transanal

irritating discharge which lasted for several weeks, producing perianal skin irritation.

3. **Perianal skin irritaion.** With frequent bowel movements, and especially of ileal contents, at least some skin irritation is to be expected during the early post-operative period. Our first case had very little. The second, the one already described, had the most. She even developed papillary thickenings of the perianal skin, and had moderate burning and itching. However, as soon as the local intrarectal infection subsided, all anal symptoms disappeared—no more pruritus, burning nor discomfort. The papillary skin thickenings, though, have persisted.

The third and last case had some irritation, but it was never severe.

In all these cases we have attempted to prevent and control the skin irritation with the use of different substances to protect the skin, as well as by avoiding any type of local rubbing or scratching. Instead of wiping the anus after each bowel movement, we have recommended washing it with water (or a sitz bath) and gently drying the area afterwards. We have aimed to keep that area as dry as possible.

The substances which we have used to protect the skin are: compound tincture of benzoin, aluminum paste, zinc stereate powder, and covicone cream, usually a combination of at least two of these.

4. **Electrolyte imbalance.** We ran into this complication in our last case, a case of chronic ulcerative colitis with intractable hemorrhagic diarrhea, who was in a very poor physical condition to start with. She had very little reserve, and it was not surprising that she did have some electrolyte disturbance. Fortunately, she responded quickly to parenteral and oral feedings.

5. **Infection.** Our second case developed an intrarectal infection, which we have already discussed.

As to the functional and general results in our three cases, we are highly satisfied. All three cases are in good physical condition and have no complaints referable to their operation. The three of them have perfect sphincter control without any involuntary leakage whatsoever. They eat a normal diet, avoiding, of course, any particular food which will tend to give them diarrhea. They gained weight after the operation, and have maintained that weight since. They are back to work, and are able to participate in the normal activities of people of their age.

F. A. C., white male, 27 years old, referred to us with diagnosis of bleeding hemorrhoids. He had had blood mixed with stools, together with some mucus, for 6 months. Stools were usually soft and had increased in frequency from 2 or 3 per day to 5 per day.

Digital rectal examination revealed the presence of multiple small polyps in the rectum. On sigmoidoscopic, it was found that both the rectum and rectosigmoid were full with small polyps. Barium enema showed the process to involve the whole colon.

A family survey revealed that the father had "suffered from polyps," and had had a colectomy with ileorectosigmoidostomy done in three stages. He had done well after that.

The patients's two sisters and three brothers were examined, and all of them were found to have polyposis.

The patient was admitted to the Presbyterian Hospital, and on 4-3-52, a one stage total colectomy with transanal ileostomy ("transanal sleeve ileostomy") was performed. He had a satisfactory post-operative course. The general state of health was excellent all the time, and the patient gained weight progressively. Yet, he was troubled by occasional crampy abdominal pains, frequent bowel movements, and perianal irritation. The latter was alleviated with 10% aluminum paste. It was never severe. The crampy abdominal pains were not frequent, and subsided in a few weeks.

The patient has been followed closely since his discharge from the hospital. He has been comfortable all the time. The perianal irritation subsided completely, and the frequency of bowel movements have decreased. Anal continence has been excellent all the time.

On his last office visit (2-5-54), almost 2 years after the operation, he was in perfect general health, with good appetite, normal weight, and no complaints. Bowels move 3 to 6 times in 24 hours. No anal irritation. Perfect sphincter control. No blood in stools. No abdominal discomfort. In short he behaves as a normal, healthy young male.

E. A. C., 13 years old white girl, youngest sister of the above discussed patient. She had no gastrointestinal complaints, but on physical, proctosigmoidoscopic and radiological examinations, was found to have polyposis, with extensive involvement of the rectal mucosa.

She was admitted to the San Juan City Hospital, and on 6-27-52, a transanal sleeve ileostomy procedure was performed. The "pulled through" terminal ileum was previously incised down to the mucosa, longitudinally at the antimesenteric border, and circularly at various levels in an attempt to reduce the number of bowel movements, as has been discussed elsewhere in this paper.

For the first 10 to 14 days post-operatively, the results were surprisingly good. She had only 3 or 4 bowel movements daily, and had normal sphincter control. Unfortunately, an abscess developed within the "rectal shell", apparently due to a slight

retraction of the ileum, which had been a little too short to start with.

As a result of the infection, the patient had, for several weeks, a frequent transanal discharge, aside from the bowel movements. Pruritic perianal irritation followed, with eventual development of flat papillary skin growths, of the condyloma accuminata type, at the anal margins.

When the intrarectal infection subsided, the perianal irritation disappeared, though the papillomata have remained.

On her last office visit, (2-8-54), 20 months after the operation, she was in excellent health, with no complaints whatsoever. She had good appetite; had no abdominal nor anal discomfort; had 2 to 4 bowel movements per day; had no blood or mucus in the stools; and had normal sphincter function.

C. G. H., 22 years old, white, female, referred to us for surgical treatment, with diagnosis of intractable chronic ulcerative colitis, with massive bleeding and secondary polyposis. Adequate medical treatment had failed to control the disease. Besides, pseudopolyposis was demonstrable on X-rays.

The patient was emaciated, weighing 60 lbs. She was prepared with transfusions, parenteral infusions, vitamins, antibiotics, etc., and on 2-5-53, a one stage transanal sleeve ileostomy was performed at the Doctor's Hospital. The dissection of the rectal mucosa was difficult, and, definitely, incomplete.

The convalescence was slow. The patient was bothered by moderately severe abdominal cramps, frequent bowel movements, and perianal irritation. Besides, she went into an electrolyte imbalance from which she recovered quickly under parenteral and oral medications.

On discharge from the hospital, she weighed 73 lbs., but felt fairly well. The abdominal cramps had subsided, and the perianal skin irritation was very slight. However, she was still having many small bowel movements per day, as much as 20, or more.

She has gradually improved since then, and, on her last office visit, on 2-19-54, she looked just like any normal young female. She is back at her work as office girl with the P. R. Telephone Co. Her weight has come up to 104 lbs., 44 lbs. over her weight at the time of operation. She has normal sphincter function and has no perianal skin irritation, but is still having many bowel movements per day (10 to 12 in 24 hours). She can hold back a movement for close to half an hour, after the initial desire, but if she tries to hold it any longer, then she has a slight leakage of liquid material.

Rectal examination, on her last visit, showed a normal appearing anus, with excellent sphincter function. The terminal ileum

was felt implanted along the anterior wall of the rectal pouch. The latter has remained as a separate entity, because its mucosa was not completely removed, as we have previously stated. That is why we now advise the removal of part of the muscular wall, with whatever remaining mucosa there might be, in cases of chronic ulcerative colitis who are to have a transanal sleeve ileostomy performed.

SUMMARY

A brief discussion of the problems created by a permanent ileostomy has been presented. This surgical procedure is indicated in some cases of diffuse polyposis, chronic ulcerative colitis, multiple carcinomas of the colon and rectum, and, possibly, in megacolon involving the whole colon.

The main complications of abdominal ileostomy are: Prolapse of the stump, retraction of the stump, gangrene of the stump, perforations with fistula formation, strictures, electrolyte imbalance, nutritional disturbances, skin irritation and ulcerations, and crampy abdominal pains.

The use of ileoproctostomy in preference to abdominal ileostomy has been discussed with the presentation of excellent results in a series of three cases, two of diffuse familial polyposis and one of chronic ulcerative colitis. The technique used has been a modification of the Ravitch procedure. We like to call this operation "transanal sleeve ileostomy".

The complications that we have encountered in the three cases were discussed. They were: crampy abdominal pain, frequent bowel movements, perianal skin irritation, intrarectal infection, and electrolyte imbalance.

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ANURIA*

*J. A. DE JESÚS, M.D.***

A sudden sharp reduction in the amount of urine excreted calls for immediate consideration of its cause. Conditions, such as obstruction of the posterior urethra and vesical neck or obstruction due to calculi or neoplasm are usually suspected from history, physical examination and catheterization. Inability to urinate may be due to neurologic disturbances, such as traumatic, inflammatory or neoplastic myelitis, but the cause in such cases should be evident. The finding of an overdistended bladder furnishes evidence that the anuria or oliguria is not due to inability of the kidneys to function properly.

The conditions just mentioned cannot be considered strictly medical and therefore will not be considered here as anuria will be discussed when it is a medical problem.

In order for the kidney to function properly certain major physiologic conditions must be fulfilled:

1. The total volume of the circulating blood must be adequate.
2. The rate and quantity of blood passing through the kidney must be adequate.
3. Hydrostatic and colloid osmotic pressures must be adequate.
4. The urinary tract must be free of obstruction.
5. The intrarenal, luminal and capsular osmotic pressures must be adequate.
6. The glomeruli and tubules must be morphologically normal.

Very seldom if ever, one encounters only one of these factors operating by itself in causing oliguria or anuria. Usually two or more factors interplay to bring about diminution or cessation of function. Therefore it seems futile to classify anuria in terms of derangement of a single physiological function. A number of medical conditions in which anuria may be encountered will be mentioned and the mechanism involved in their production discussed.

I. **Shock:**

Whether shock is produced by peripheral circulatory collapse or by loss of blood volume makes no difference in so far as the

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production of anuria is concerned. The sequence of events is as follows: First, there is a prolonged fall in blood pressure followed by a reduction of blood volume. The reduction of blood volume may be actual or relative; actual, from blood loss or extravasation of plasma or blood into tissues; relative in peripheral vascular collapse where the blood volume is small in relation to the size of the vascular bed. This causes a reduction in renal blood flow which in turn causes renal ischemia with damage of the tubules. During the collapse, the blood pressure is so low that ischemia results. Then the function of the glomeruli is altered, as evidenced by the presence of protein in the glomerular capsular space. In spite of this functional damage, histological changes of the tufts other than anemia of the glomeruli, are absent. Since the blood supply to the tubules is derived from the glomerular blood flow, the insufficient perfusion of the glomeruli leads to progressive necrobiotic changes of the convoluted tubules.

The tubular lesion is purely epithelial in character and, as in all epithelial lesions, a complete recovery without scar formation is possible. On the other hand, when the glomeruli, which are derived from the mesenchyma, are involved, complete regeneration is much less probable, because scar formation and fibrous degeneration of the glomeruli will result. Regeneration of the tubules starts after an average of 10 days. Thus in most cases of lower nephron nephrosis, spontaneous diuresis sets in after 10 days and the seemingly fatally ill patients recover.

It was thought that blocking of the tubules by necrotic material stopped the urinary flow. This explanation is, at best, only partially correct. It is much more probable that the damaged tubular walls loose their specific reabsortive power which is present in the normal tubule. In lower nephron nephrosis, the glomeruli form an ultrafiltrate of the blood plasma in the normal way. During the passage through the damaged or necrotic tubules, all the glomerular filtrate is absorbed because the tubular epithelium has been transformed into a nonspecific dialyzing membrane. The colloidal osmotic pressure of the circulating plasma proteins draws all the water present in the lumen of the necrotic tubules into the blood stream with resulting anuria.

II. **Dehydration:**

The oliguria which accompanies severe dehydration is initially a physiologic response. A major function of the kidney is to maintain fluid balance. In the presence of severe dehydration, the kidney becomes frugal with the scanty fluids available within the body. Extremely small amounts of urine of high specific

gravity are eliminated by the normal kidney until fluids become available again. When water and electrolytes are lost because of severe diarrhea, excessive vomiting, profuse sweating, or a combination of these, and neither water nor electrolyte is administered to replenish the losses, the following physiological factors will interplay to produce anuria:

a. The circulating blood volume diminishes and becomes inadequate for proper renal circulation.
b. The osmotic pressure in blood increases thus diminishing the head of pressure between hydrostatic and osmotic pressure, an important factor in effective filtration pressure.
c. The increased osmotic pressure of blood will stimulate the osmo-receptor which in turn will stimulate the pituitary to produce the antidiuretic hormone.
d. The increased osmotic pressure of blood enhances tubular reabsorption.

If dehydration goes unchecked the blood volume diminishes to the point at which the renal blood flow is compromised to such an extent that ischemia of the tubules will result. Damage indistinguishable from the one described under shock takes place, the anuria then becoming unresponsive to hydration and electrolyte replacement. At this stage the patient is suffering from a lower nephron nephrosis.

III. Post-Operative Anuria:

Post-operative anuria is of common occurrence. During surgical procedures, especially if the operation is extensive, the loss of blood, the injury of tissues, the prolonged anesthesia and the dehydratation from sweating can cause severe hypotension and anuria. Under conditions of hypotension and reduced renal blood flow, the effective filtration pressure is insufficient for elaboration of significant quantities of glomerular filtrate. In the presence of hemoconcentration most of the filtrate is reabsorbed by the renal tubular epithelium and anuria is the outcome. If this state of hypotension and dehydration is not corrected, the renal tubule will suffer from ischemia and the serious problem described under shock and dehydration will ensue.

IV. Poisoning:

In cases of anuria due to poisoning whether from heavy metals or from other drugs such as the sulfonamides, the renal tubules undergo damage. As the glomerular filtrate is concentrated in the

tubules the concentration of the noxious agent (metal or drug) reaches a level which will injure the tubular cell in a way histologically similar to that seen in shock. Moreover many of these patients either go into shock or have severe diarrhea or severe vomiting in addition. Thus the factors of collapse and dehydration causing ischemia as well as the direct damage of the tubule by the poison play an important role in the anuria as seen in these patients.

V. Mismatched Transfusions:

It has been stated on many occasions that the anuria consequent to transfusion reactions is due to plugging of the renal tubules by pigment casts. There is experimental evidence which tends to support this. Baker and Dodds injected rabbits with hemoglobin and its derivates. The pigment precipitated and blocked the renal tubules, the rabbits becoming anuric. There are scores of similar experiments in rats and dogs with similar results. When each of the experiments is repeated under controlled conditions (that is control of dehydration, shock, etc.) no anuria results. If animals are acidotic, dehydrated, or tubules are injured by poisoning, the pigments then are able to do the damage. Renal ischemia also enhances the precipitation of pigment casts and anuria. Cannan and Redish have injected hemoglobin in dose of .64 gm/kg. in man without adverse effects.

The evidence indicates that all the pigments tried (except for hematin) are innocuous in normal animals with respect to both the systemic circulations and the kidneys. Acidification of the urine is not sufficient to cause protein precipitation, invariably, and when it does one may suspect the presence of one or more additional factors. Casts have been produced experimentally most consistently in rabbits, in which the sympathetic system is highly labile. Rabbits are known to be highly sensitive to psychogenic renal vasoconstriction.

Anoxia is the cornerstone of anuria due to tubular damage. Other insults, such as chemical intoxication, allergy, dehydration, etc., may substantially aid in the production of tubular damage.

VI. Pyelonephritis and Glomerulonephritis:

Anuria is occasionally seen in these conditions. More often one sees oliguria. No adequate explanation has been given for this phenomenon other than the fact that there is glomerular damage of sufficient magnitude to cause circulatory disturbances in the tubules and that local circulatory disturbances in the glomeruli will affect filtration.

TREATMENT OF ANURIA

Prophylaxis:

During the first stage of the treatment, that of prophylaxis every effort should be made to keep patients from going into shock and once they are in shock, energetic treatment for shock should be instituted.

If severe dehydration is recognized early and the lost fluids and electrolytes are judiciously replaced, normal kidneys can handle the situation easily. These are the clearcut cases of clinically encountered anuria in which the patients are cured permanently simply by judicious treatment of their dehydration. The clinical problem in the presence of anuria of dehydration is one of fluid and electrolyte balance, it is not a renal problem.

If postoperative circulatory collapse is combated early by intravenous administration of compatible fresh blood and an appropriate concentration and quantity of electrolytes, the kidneys will remain normal and the anuria soon will be alleviated. If the postoperative state of shock endures, renal damage may set in and the anuria may become more severe.

In poisoning, the treatment of the acute emergency varies in accordance with the agent ingested. Shock and dehydration must be combated if present.

In mismatched transfusions prophylaxis consists mainly in careful cross-matching of the blood and in checking and rechecking to see that the blood to be transfused has actually being crossmatched with the proper recipient and not with another patient with the same or similar name. Transfusion should be discontinued at the first sign of reaction and the following steps taken:

1. Check serum for hemolysis and urine for hemoglobinuria as they will appear almost instantaneously. This can be done while the patient is under anesthesia and there is a sudden unexplainable drop in blood pressure or any other unexplainable phenomena. The infusion can be switched over immediately to some other fluid while the serum and urine (if available) are tested as well as recrossmatching.

2. If transfusion reaction is confirmed or strongly suspected one is justified in giving 50 cc $NaHCO_3$ 5 - 7% I.V. over a period of 5 minutes. Slow down if tetanic twitching or circumoral pallor is noted.

3. 10% glucose in water I.V. as rapidly as possible 2,000 cc in 1½ hours.

4. Combat shock.

5. If diuresis starts soon, continue dextrose in water for 8-12 hours.

6. If diuresis does not set in — conservative restricted fluid regime is in order as outlined under the third stage of treatment.

The amount of water lost through respiration and perspiration should be replaced in addition to losses from vomiting and diarrhea. The amount usually lost through the skin and lungs is 800-1000 cc per day. No protein should be given because the nitrogenous metabolites such as urea, uric acid and creatinine cannot be eliminated. It has been stressed that complete starvation leads to catabolism of body proteins and that this will even increase the nitrogenous products retained in blood and body fluids. Moreover, potassium, which is an important factor in uremia, is also liberated during the endogenous breakdown of proteins. Finally, in many cases of acute anuria, the function of the liver is involved and the total protein content of the serum falls below normal limits. To combat the catabolism of body proteins and also hypoproteinemia, administration of proteins to patients with acute anuria has been advocated. Weighing the danger of the nitrogen retention due to ingestion of proteins to anuric patients against the danger of catabolism of proteins during starvation, the first is certainly more dangerous than the second. For all practical purposes, administration of calories in the form of carbohydrates and fats which are oxidized to $CO_2 + H_2O$ reduces the catabolism of protein during hunger. Thus it may be advisable to add sugar to the daily ration of 800 - 1000 cc of water.

Borst has advocated giving in addition every day a mixture of 200 gm. of butter and 200 gm. of sugar but in most patients with acute anuria, this umpalatable mixture causes nausea and vomiting.

No patient with acute anuria can be treated without careful analysis of the serum. Determinations of: blood urea nitrogen, non-protein nitrogen, creatinine, uric acid, chlorides, sodium, potassium and CO_2 combining power become imperative.

As most patients with acute anuria are in shock, their serum chloride is usually low. Also their serum sodium is often low. When hypochloremia is present, sufficient amounts of sodium chloride must be added to the daily ration of 800 - 1000 cc of fluid. In those cases the glucose is given in saline.

In patients with acidosis who are not nauseated $NaHCO_3$ can be given by mouth (2-3 teaspoonfuls per day). In most cases alkali will have to be given parenterally. Intravenous M/6 Na-lactate is not advisable because this requires introduction of larger amounts of fluid which will compromise the circulation and lead to left heart failure. Preferable is the intravenous administration

of 5% $NaHCO_3$ solution. The potassium content of the blood and body fluids has a tendency to increase. Hyperpotassemia has an unfavorable influence on the heart. EKG signs of cardiac damage often develop when the potassium content of the serum rises above 6.4 mEq/L. When the potassium exceeds 7.4 mEq/L the outlook is serious. Fortunately, the increase of the blood K in patients with acute anuria treated with starvation is only moderate and hardly ever requires treatment. Rare cases have been described where, during anuria, the K level is decreased.

In cases where the potassium climbs to dangerous levels, intestinal irrigation is useful and even imperative in the absence of an artificial kidney or peritoneal dialysis. It is a relatively simple procedure and can be performed in institutions where the artificial kidney is not available. Another measure for lowering serum K is the administration of insulin with the glucose as the deposition of glucose into the cell is accompanied by migration of K into the cells.

Diuretic or recovery stage, that of diuresis, dehydration and negative electrolyte balance may occur suddenly. The fluid and electrolyte requirements of a patient should be carefully followed and satisfied at all times. This should take into account loss through urine, stool, vomitus and insensible perspiration.

In pyelonephritic and glomerulonephritic anuria the treatment is that of the primary condition. Watchful expectancy is best.

SUMMARY

Some of the medical conditions in which anuria may be encountered have been mentioned and the mechanism of renal shut down described. The treatment of these conditions has been outlined in three stages.

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VALORES NORMALES DE 17-CETOESTEROIDES URINARIOS EN PUERTO RICO*

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Las determinaciones de 17-cetoesteroides urinarios en el diagnóstico de distintas condiciones patológicas, han despertado un creciente interés en la clase médica de Puerto Rico. Sin embargo, debido a que los métodos más conocidos resultaban muy largos y costosos, la mayoría de los laboratorios de la Isla, no estaban haciendo estas determinaciones.

El hecho de que Friedman[1] informó una disminución en la excreción de 17-cetoesteroides en Indios varones y el hecho de que existen factores climatológicos, raciales o étnicos, dietéticos, etc., que pueden afectar la eliminación urinaria de estos esteroides, nos indujo a estudiar el problema en individuos sanos puertorriqueños para establecer los valores normales en nuestro medio ambiente.

Los métodos más usados para estas determinaciones consisten, principalmente de las siguientes etapas:

(a) Hidrólisis de la orina.
(b) Extracción de las substancias cetoesteroideas.
(c) Evaporación del líquido usado para la extracción, y
(d) La reacción del color, principalmente por el método de Zimmerman.

Fister[2] en su manual de "**Procedimiento para Química Espectrofotométrica**," da un método para la determinación total de 17-cetoesteroides basado en procedimientos y modificaciones de Zimmerman, Giralt, Holtorff y Kock, Callow y Emmens, y otros que no es necesario mencionar en este trabajo. Este método es uno de los más confiables usados, pero indudablemente demasiado largo y costoso para el laboratorio promedio.

Existen, sin embargo, modificaciones a este procedimiento que, sin incurrir en menoscabo de la seguridad de los resultados, requieren equipo menos costoso, más fácil de manipular y disminuyen considerablemente el tiempo requerido para la prueba.

Hemos utilizado para el estudio que presentamos en esta ocasión el método de Drekter[3] y colaboradores del New York Medical College. Estos, enfrentados con la necesidad de determinar 17-cetoesteroides urinarios durante un estudio que debían hacer, viéronse obligados a adoptar un método basado en el ya mencionado por Fister,[2] pero más corto y que requiriese menos cantidad de orina, dando resultados igualmente confiables.

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Para mayor seguridad en los resultados que habríamos de obtener en nuestro estudio, hicimos paralelamente varias determinaciones de 17-cetoesteroides por el método según aparece en el Manual de Fister y por el método más corto de Drekter y sus colaboradores. Los resultados obtenidos fueron satisfactorios y creímos justificada la adopción del segundo método para la presente investigación.

Queremos hacer mención al hecho de que, conscientes en todo momento de estar trabajando con un método cuyos resultados se calculan a base de muestras de orina de 24 horas, pusimos especial cuidado en la selección de personas conscientes y responsables.

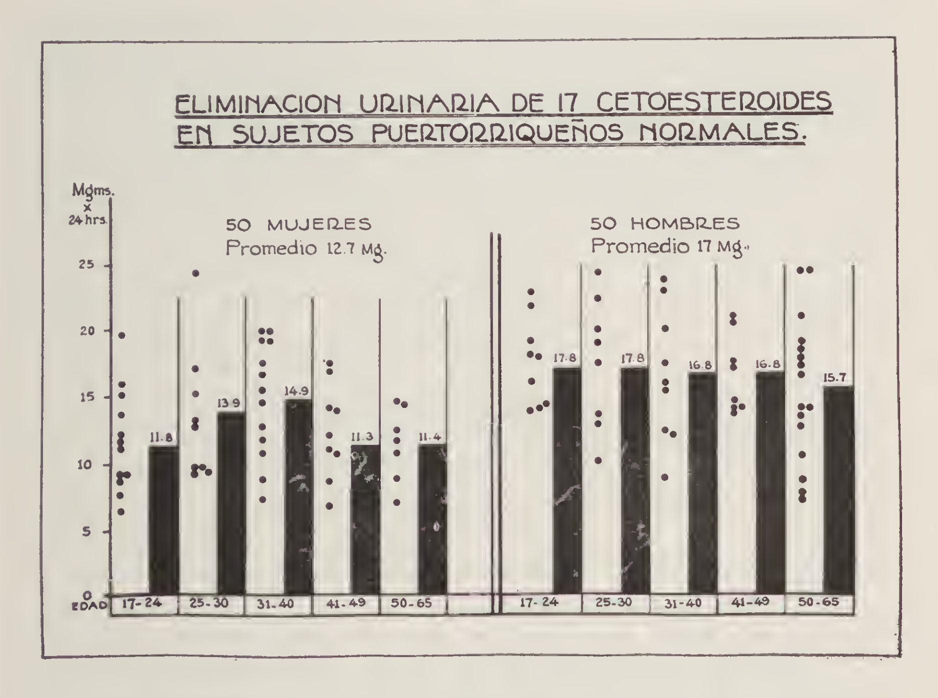
RESULTADOS

Hicimos determinaciones de 17-cetoesteroides en un grupo de 50 mujeres y 50 hombres, todas personas aparentemente normales y saludables. Tuvimos, además cuidado en que ninguna de estas personas estuviera bajo tratamiento de hormonas que pudieran afectar la excreción de los 17-cetoesteroides.

La siguiente Gráfica expresa los resultados obtenidos durante esta investigación. Con el objeto de obtener información adicional, dividimos cada grupo de 5 sub-grupos de acuerdo con las edades. En la Gráfica, las barras sólidas representan el promedio de la eliminación de 17-cetoesteroides en milígramos en 24 horas. Así la eliminación obtenida en el grupo de mujeres de 17 a 24 años, varió entre 6.38 mgs. y 19.9 mgs. con un promedio de 11.8 mgs. En el de 25 a 30 años, la eliminación mínima fué de 9.5 mgs. y la máxima 24.5 mgs., con un promedio de 13.9 mgs. En el de 31 a 40 años, varió entre 7.2 mgs., y 20.0 mgs., con un promedio de 14.9 mgs. En el de 41 a 49 años, la eliminación mínima fué de 6.7 mgs., y la máxima de 17.5 mgs., con un promedio de 11.3 mgs., y en el de 50 a 65 años la mínima varió entre 7.0 mgs. y 14.7 mgs., siendo el promedio 11.4 mgs. A la izquierda de cada barra aparecen los puntos que representan los valores obtenidos en cada grupo.

El promedio total de las 50 determinaciones en las mujeres fué de 12.7 mgs.

Aunque comprendemos que no podemos llegar a una conclusión definitiva en cuanto a la relación entre la edad y la eliminación de los 17-cetoesteroides, la Gráfica sugiere un ligero aumento en la eliminación en el caso de las mujeres hasta la edad de 40 años más o menos, después de lo cual, hay una disminución aparente entre los 40 y 50 años, manteniéndose este nivel más o menos constante, aún hasta los 65 años, que es la edad mayor comprendida en este estudio.



La segunda parte de la Gráfica expresa a su vez los valores obtenidos en hombres, divididos los grupos por edades. En este caso en el grupo de hombres de 17 a 24 años, la eliminación mínima obtenida fué 14.0 mgs. y la máxima 23.0 mgs., con un promedio de excreción total de 17.8 mgs. En el grupo de 25 a 30 años, la excreción mínima fué 10.1 mgs., la máxima 24.5 mgs. con un promedio de 17.8 mgs. En el de 31 a 40 años, la excreción mínima fué 9.0 mgs., la máxima 24.1, y el promedio 16.8 mgs. En el de 41 a 49, la excreción mínima fué 13.9 mgs., la máxima 21.2 mgs., y el promedio fué 16.8 mgs. En el de 50 a 65 años, la excreción varió entre 7.8 mgs. y 24.8 mgs., con un promedio de 15.7 mgs.

El promedio total de las 50 determinaciones en hombres fué de 17 mgs.

Aunque hay muy poca diferencia entre la eliminación de los distintos grupos de hombres, es evidente que la excreción máxima se observa en los primeros dos sub-grupos. Solamente en el último sub-grupo hay una disminución sugestiva de que durante estas edades, la excreción en el hombre empieza a disminuir visiblemente.

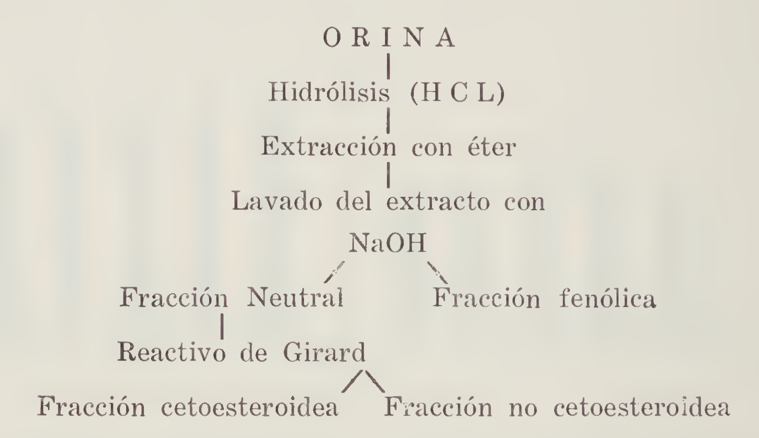
DISCUSION

Es de gran importancia comparar nuestros resultados con otros obtenidos en investigaciones similares a ésta y llevadas a cabo en otros países.

Antes de entrar en estas consideraciones, sin embargo, vale la

pena, llamar la atención hacia ciertos aspectos de estos trabajos que arrojan luz y ayudan a una mejor comprensión de las diferencias, muchas veces considerables, que encontramos entre los valores obtenidos por nosotros y los de otros investigadores.

El siguiente esquema representa las distintas etapas a que se somete la orina y sus derivados durante la determinación cuantitativa de los 17-cetoesteroides.



Vemos que la orina se somete a un proceso de hidrólisis con ácido clorhídrico concentrado (HCl). Luego se lleva a cabo la extracción con éter; se lava este extracto con hidróxido de sodio (NaOH) eliminando así las substancias fenólicas, como la estrona, ácidos, y algunos pigmentos extraídos de la orina, quedando disueltos en el éter la fracción neutral que contiene los 17-cetoesteroides y otras substancias no cetoesteroideas.

Hacemos mención de este esquema para llamar la atención al hecho de que en nuestro estudio hicimos la determinación de los 17-cetoesteroides en esa fracción neutral, a pesar de que por el método del reactivo de Girald, es posible purificar más este extracto antes de someterlo a la reacción de Zimmerman, obteniendo así valores más exactos.

Creemos, sin embargo, que la purificación del extracto por el método de Girald, no justifica el trabajo adicional que ello conlleva. Nos parece que desde el punto de vista de diagnóstico clínico, no es necesario una exactitud tan exagerada si se tiene en cuenta que la fracción de las substancias no cetoesteroideas representan un porciento del total, que no es lo suficientemente alto para introducir confusión en la interpretación de los resultados.

Las observaciones arriba mencionadas nos decidieron a hacer nuestras determinaciones en el extracto neutral que incluye los 17-cetoesteroides al igual que otras substancias no cetoesteroideas. Drekter[3] y sus colaboradores, a los cuales ya nos hemos referido,

no emplean estas separaciones; tampoco la emplea Escamilla[4] en el método que da en su libro "Laboratory Aids in Endocrine Diagnosis", cuyo método es muy parecido al usado por nosotros.

Con estas consideraciones en mente, pasemos ahora a examinar valores obtenidos por otros investigadores en individuos normales.

Escamilla[4] de la Escuela de Medicina de la Universidad de California, en su libro ya mencionado, da para hombres una variación normal de 10 a 20 mgs., con un promedio de 15 mgs. en 24 horas, y en las mujeres, una variación de 10 a 15 mgs. con un promedio de 10 mgs. en 24 horas.

Fister[2] en su Manual, da para hombres un promedio diario aproximadamente de 17 mgs. y para mujeres de 13 mgs. diarios en el extracto no purificado.

Barnett[5] y colaboradores, obtuvieron un resultado de 18.4 mgs. para los hombres y 13.2 para las mujeres.

Harold L. Mayson, de la Fundación Mayo, y William Engstrom[6] de la Universidad de Yale, en un estudio relacionado con 17-cetoesteroides, citan los siguientes promedios de eliminación obtenidos en distintos grupos por distintos investigadores: Hombres de 17 a 30 años, 14.1 mgs.; de 20 a 40, 13.6 mgs.; de 17 a 34, 18.0 mgs. Mujeres de 20 a 35 años, 10.0 mgs; de 20 a 40, 7.6 mgs; de 21 a 45 años, 11.6 mgs.

Friedman[1] encontró que la excreción en 23 nativos del Sur de la India varió entre 7.05 y 17.1 mgs.; en 13 sujetos del Norte, los 17-cetoesteroides dieron una cifra mínima de 7.9 y máxima de 14.04 mgs., mientras que en 15 Europeos, las cifras variaron entre 12.1 y 31.1 mgs.

Finalmente, la Dra. Angel[7] y sus colaboradores del Hospital de Enfermedades de la Nutrición en México, informaron en adultos, valores entre 4 y 13.8 mgs., siendo el promedio en las mujeres, 7.9 mgs. En los hombres encontraron un promedio de 11.2 mgs. variando entre 6.8 y 22.4 mgs. en 24 horas. En niños menores de 15 años, estos autores encontraron valores entre 1.4 a 5.3 mgs. con un promedio de 3.6 mgs. en las niñas. En los varones encontraron valores de 0.7 a 7.3 mgs. con promedio de 4.4 mgs.

Los valores arriba expuestos son solamente representativos de los muchos trabajos que en este campo de investigación se han hecho y es evidente que existen discrepancias marcadas entre las cifras obtenidas por unos y por otros, aunque se hayan obtenido con métodos distintos. Esta evidencia justifica pues, que una vez una institución haya adoptado un método, es aconsejable la determinación de los valores normales a obtenerse por dicho método.

CONCLUSION

Los valores normales de 17-cetoesteroides urinarios en Puerto Rico son para mujeres 12.7 mgs.; para hombres 17 mgs./24 horas, siendo éstos iguales a los informados por investigadores norteamericanos para los Estados Unidos continentales.

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ANEURYSM OF THE RENAL ARTERY

REPORT OF ONE CASE

*PABLO G. CURBELO, M.D.**

Aneurysm of the renal artery is a rare lesion which is difficult to diagnose at times. Moreover, the symptoms thereof may mimic very closely those of almost all of the acute surgical emergencies of the abdomen. In these circumstances, clinical problems related to this condition are of interest to general surgeons, urologists and obstetricians alike.

Symptoms

Prior to rupture, most true aneurysms are devoid of symptoms, except for vague gastro-intestinal discomfort. Whenever symptoms are present, however, they are usually due to some associated urological condition. After rupture has taken place, the patient exhibits varying degrees of pain in the side, the symptoms and signs of shock and acute hemorrhage and usually a mass in the flank. Hematuria also occurs occasionally.

Diagnosis

Calcification in the wall of the aneurysm is present in 50% of the cases. It casts a characteristic roentgen shadow, to wit: it is round or oval in shape, has a clear center, and may resemble a signet ring. The shadow is extra-renal and may cause a compression-filling defect in the pelvis. In the lateral film, the shadow appears on a plane anterior to the pelvis. The lesion is most clearly demonstrated by arteriography and it is quite probable that the incidence of aneurysm will rise as this method comes into more general use.

False aneurysms appear as a rather indefinite blurring on the affected side in the x-ray film.

From the standpoint of the clinical recognition of this condition prior to rupture of the vascular wall, the important factor is to be aware that this condition may be responsible for an obscure abdominal condition characterized by pain, shock, and acute anemia.

Differential Diagnosis

The unruptured true aneurysm may be confused by the radiologist with a calcified lymphatic gland, cyst, or abscess. The diagnosis of ruptured peptic ulcer, acute intestinal obstruction, and acute hemorrhagic pancreatitis have been made at times when the condition was actually a ruptured renal aneurysm. Similarly,

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* From the Bayamón Charity District Hospital.

the obstetrician has been misled into making the diagnosis of ruptured ectopic pregnancy, ruptured abdominal pregnancy, and premature separation of the placenta. Urological conditions with which aneurysm of the renal artery may be confused include renal cyst, hydronephrosis, perinephric abscess, and necrotizing renal tumor.

Hack has collected from the literature five instances of rupture of the renal artery during pregnancy and added two of his own. The symptoms in each patient were very similar: abdominal pain, shock, and acute anemia. The train of symptoms occurred on two separate occasions, the diagnosis was not made and all of the patients died.

Salmon has reported the case of a 13 year-old girl who showed left lumbar pain and frequency of urination on admission. An intravenous pyelogram disclosed an absence of function on the left side and the diagnosis of left renal tuberculosis was made in the light of previous evidence of Pott's disease. The patient was placed under streptomycin therapy; but the attacks of abdominal pain persisted. The psychiatric consultation was in favor of a psychogenic nature of the pain. After two months, a mass appeared in the left flank and the diagnosis of left perinephric abscess was made. At operation the muscles and the dorsal fascia were found to be ecchymotic which is the Uhle sign of ruptured renal aneurysm. However, the significance of this sign was overlooked and on incising the fascia massive uncontrollable hemorrhage took place. Baker and Huckelberry reported a case of ruptured intrarenal aneurysm clinically diagnosed as a renal tumor and in which the true diagnosis was not made until the specimen was examined by the pathologist.

CASE REPORT

D. de H., Bayamón District Hospital No. 30907, female, 33 years old.

On her first admission on December 21, 1943 her symptoms appeared to be gastro-intestinal in nature, to wit: post-prandial pain and fullness in epigastrium and pain in left lumbar region. At that time, the only positive diagnosis was that of uncinariasis, which was treated properly.

The patient disappeared from view until September 20, 1951 when she was re-admitted to the Hospital. The interval history was that her gastro-intestinal symptoms had persisted in spite of treatment by various outside physicians. A gastro-intestinal series showed no evidence of pathology, but a calcific shadow in the right upper quadrant was diagnosed as a probable gallstone. The patient left the hospital slightly improved from her digestive symptoms.

On February 4,1952 I saw the patient for the first time. The epigastric and left lumbar pain was now associated with some frequency and dysuria. An intravenous pyelogram showed an irregularly shaped urinary calculus in the left renal pelvis, and a calcification on the right side which lied outside the urinary tract and had the appearance of a gallstone.

On April 17 the calculus from the left renal pelvis was removed by pelviotomy and the patient was discharged from the hospital on May 2.

However, her digestive symptoms persisted, and on February 3, 1953 another gastro-intestinal series failed to turn up anything of clinical importance. Gallbladder studies were also entirely negative. At that time patient began having episodes of left pyelonephritis with unsatisfactory response to chemotherapy and antibiotics. A re-check intravenous pyelogram revealed signs of left pyelonephritis and the probability of an aneurysm of the right renal artery was seriously considered. On April 19, 1954 an aortogram offered incontrovertible evidence of the presence of a true aneurysm of the main right renal artery.

Digestive symptoms as well as episodes of pyelonephritis persisted and on February 24, 1955 left nephrectomy for atrophic pyelonephritis was carried out. The patient had an uneventful recovery. Her digestive disturbances have greatly diminished and no further episodes of pyelonephritis have taken place; the patient has gained some weight and feels and looks much better than before the operation.



Fig. 1
Intravenous pyelogram. Typical roentgen picture of right renal aneurysm.

Fig. 2
Aortogram showing aneurysm of main right renal artery.

DISCUSSION

At autopsy, the incidence of aneurysm of the renal artery is about one in thirteen thousand. The clinical incidence is much lower: less than one hundred cases have been reported in the literature. One per cent of all aneurysms are located in the renal artery.

It is customary to classify aneurysms into true and false types. A true aneurysm is one in which the sac is formed by the walls of the artery; while a false aneurysm is one in which the walls are formed by the tissues surroundnig the area of rupture of an artery or an aneurysm which has ruptured.

True aneurysms are due to some congenital weakness or disease of the media; while false aneurysms may result from the partial rupture of a true aneurysm or by surgical or accidental injury to the outer coats of an artery. Aneurysm of the renal artery was first described in France in 1853 from autopsy material. But it was not until 1917 that the first radiological diagnosis was made in Stockholm.

The average age at which it occurs is 47.5 years, but it may apparently occur at any age, having been reported at 2 and 82 years.

Most true aneurysms measure around 1.8 cm. in diameter and rarely reach the size of 7 cm before rupture takes place. False aneurysms, on the other hand may grow big, sometimes occupying one half of the entire abdomen. As to treatment it may be said that small calcified aneurysms which are asymptomatic may be treated by watchful waiting; but if pain develops, or if the aneurysm increases in size as shown by periodic x-ray studies, nephrectomy would be indicated provided the other kidney is capable of sustaining the load.

False aneurysms, on the other hand, demand prompt surgery because the mortality rises from 20% in patients operated to 100% in those treated expectantly. An approach which permits ligation of the artery near the aorta early in the procedure should be used. Uhle's sign should never be disregarded.

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RESECCIÓN ESOFÁGICA CON RESTABLECIMIENTO DE LA CONTINUIDAD POR MEDIO DE UN TUBO PLÁSTICO*

J. COSTAS DURIEUX, M.D.
Ponce, Puerto Rico.

Es innegable que el tratamiento ideal para un tumor o estrechez del esófago sería la resección con anastomosis directo término-terminal. Es un hecho probado que este tipo de anastomosis es imposible cuando la resección esofágica es de más de 4 cm. de largo. En muy raras ocasiones la lesión permite una resección tan pequeña. Para resolver este problema se han usado diferentes métodos. Entre los usados con más éxito se puede mencionar el uso de un asa yeyunal,[1] el uso del colon transverso[2] y por último la movilización del estómago[3, 4] dentro de la cavidad torácica. Todos estos procedimientos constituyen operaciones muy serias, chocantes y de relativa larga duración.

Estas consideraciones y el advenimiento de los plásticos con la evidencia experimental de su innocuidad dentro del cuerpo humano impulsaron a Berman a ensayar experimentalmente un tubo de polietileno para restablecer la continuidad esofágica.[7] En los animales sacrificados a distintos intervalos él encontró la formación de tejido alrededor del tubo variando desde una fina vaina gelatinoide a los cuatro días hasta la formación de una capa esofágica de 4 mm. de espesor. Esta se confundía con ambas extremidades esofágicas normales y estaba cubierta con epitelio. Alentado por las experiencias experimentales en noviembre de 1950, Berman le colocó un tubo plástico a un hombre de 75 años de edad con un carcinoma del tercio inferior del esófago y el resultado fué bueno.[8] De aquí en adelante nuevas experiencias clínicas también tuvieron éxito.[9]

Nosotros tuvimos la oportunidad de usar este procedimiento en una paciente con un carcinoma del tercio medio del esófago. La paciente estaba en condiciones físicas muy pobres y por lo tanto consideramos que no toleraría la intervención convencional.

INFORME DEL CASO

E. L. C., mujer de 56 años de edad fué admitida a la Clínica del Dr. Pila el 2 de septiembre de 1954 con historia de dolor y ardor retroesternal desde hacía cuatro años. El dolor y ardor cambió a disfagia para los alimentos sólidos y esto continuó progresando hasta establecerse alguna disfagia aun para los líquidos.

* Trabajo presentado en la Asamblea Anual de la Asociación Médica de Puerto Rico, el 9 de diciembre de 1954.

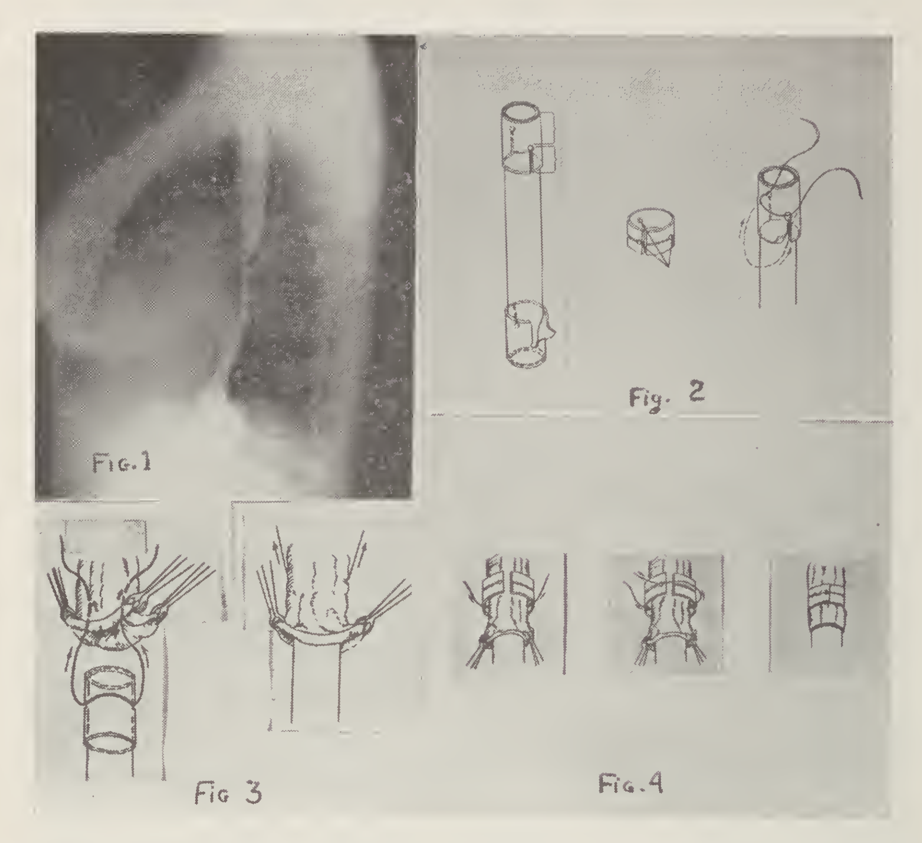


Fig. 1. Esofagograma que demuestra el tumor del esófago justamente debajo del arco aórtico.
Fig. 2. Tubo de polietileno de Berman con el anillo fijador y con la sutura en posición.
Fig. 3. Técnica para empezar a colocar el tubo.
Fig. 4. Técnica que demuestra el tubo colocado y el anillo amarrado.

En el momento de ser admitida al hospital la disfagia tenía más de seis meses de duración.

El examen físico reveló evidencias de gran pérdida de peso y de caquexia. La auscultación cardíaca arrojó la presencia de un soplo sistólico en la base. El ritmo no era normal, existiendo varios extrasístoles. El resto del examen no reveló ningún otro signo positivo.

La radiografía simple del tórax nos enseñó campos pulmonares normales en ambos lados. La silueta cardíaca estaba aumentada hacia la izquierda. El esofagograma reveló una obstrucción al nivel del arco aórtico.

El electrocardiograma mostró una hipertrofia ventricular derecha y una conducción defectuosa y prolongada.

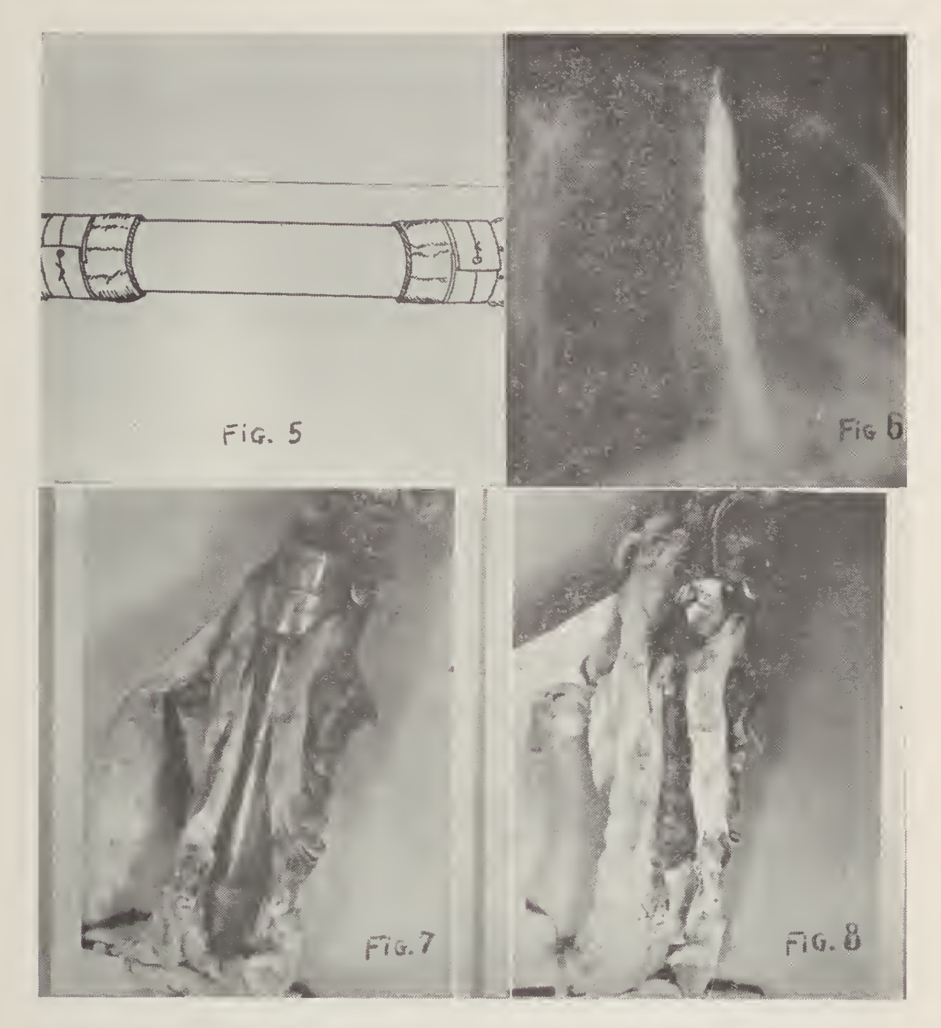


Fig. 5. Se ha terminado de restablecer la continuidad esofágica por medio del tubo.

Fig. 6. Esofagograma a los 10 días que demuestra el tubo en perfecta posición.

Fig. 7. Pieza patológica que demuestra el tubo dentro del canal recientemente formado.

Fig. 8. Pieza patológica que demuestra el nuevo canal con el tubo removido. Puede verse el epitelio a ambos extremos.

Al hacer la esofagoscopía se encontró un tumor a 20 cm. de la arcada dentaria superior.

El hemograma fué como sigue: 3,480,000 hematíes, 7,400 leucocitos y 10 gm. de hemoglobina. Los damás exámenes de laboratorio pertinentes fueron negativos.

El tratamiento preoperatorio consistió de sueros glucosados

y amigen por vía intravenosa, extracto hepático y vitaminas múltiples. También la paciente recibió 500 cc de sangre.

El 30 de septiembre de 1954 bajo anestesia endotraqueal de pentotal sódico, oxígeno y éter se practicó la operación. Con la paciente en decúbito lateral izquierdo se hizo una toracotomía derecha. Se hizo una resección de la séptima costilla y se entró al hemitórax derecho a través del lecho de dicha costilla. Se retrajo el pulmón anteriormente y se dividió la pleura mediastínica desde el cardia hasta más arriba de la vena azygos. Esta vena no fué dividida. Se encontró un tumor del esófago localizado justamente debajo de la vena azygos. No fué difícil liberar el esófago y una vez liberado fué dividido por arriba y por debajo del tumor. El esófago resecado tenía una longitud de 6 cm.

Colocamos un tubo plástico de 15 cm. de largo primero en el muñón próximo y después en el distal. El déficit cubierto tenía 9 cm. de longitud. Suturamos la pleura mediastínica por encima del tubo y se reexpandió el pulmón. Cerramos el hemitórax en la forma usual estableciendo drenaje bajo agua. Inmediatamente después hicimos una gastrostomía de tipo Stamm. La paciente toleró la operación muy bien.

El diagnóstico patológico fué carcinoma epidermoide del esófago, grado I con metástasis a ganglios regionales.

Después de la operación se conectó el tubo de la gastrostomía a una succión de Wangensteen por espacio de tres días. Se administró penicilina con estreptomicina por vía intramuscular y líquidos por vía intravenosa.

El tercer día se empezó la alimentación por el tubo de la gastrostomía. El quinto día se empezó a administrar líquidos por vía oral. La alimentación por vía oral se aumentó gradualmente y se descontinuó la alimentación por la gastrostomía.

El octavo día se le permitió a la paciente tener comidas blandas.

El décimo día se le hizo un esofagrama, pudiéndose visualizar el tubo plástico que aparecía en perfecta posición y no existía ninguna obstrucción al paso del bario.

El día onceno la paciente tuvo un mareo súbito, se puso fría y empezó a sudar copiosamente. Al examinarla encontramos una taquicardia supraventricular que más tarde cambió a fibrilación auricular. Se hizo una digitalización rápida. Se usó también quinidina y el ritmo cambió a normal al siguiente día. De aquí en adelante la paciente siguió bien. Fué dada de alta del hospital en buenas condiciones y tolerando bien una dieta blanda.

A los veinticinco días de la operación estando en su casa la paciente tuvo tos con expectoración sanguinolenta y murió súbitamente.

Se le practicó un examen post mortem encontrándose el tubo plástico en perfecta posición. Había sido cubierto totalmente por una capa gruesa de 4 mm de espesor de tejido fibroso y el epitelio esofágico había empezado a crecer a ambos extremos.

COMENTARIO

Los resultados obtenidos en este caso tienden a demostrar que la hipótesis levantada experimentalmente por Berman es correcta.

La operación fué menos traumática y menos prolongada que el procedimiento convencional.

No hubo dificultades al restablecer la continuidad por medio del tubo y el curso post operatorio en lo que a la intervención se refiere estuvo libre de complicaciones.

No hubo evidencias de obstrucción alguna. No pretendemos recomendar esta intervención para substituir las otras convencionales pero creemos que es de valor y merece probarse más.

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THE "DOOR TO DOOR" BLOOD TESTING PROJECT IN PUERTO RICO*

*ERNESTO QUINTERO, M.D.***

In Puerto Rico we have continued to lower the reservoir of syphilis in our population by the use of "door to door" mass blood testing. We have covered the large population centers of the Island, including most of the San Juan metropolitan area, **twice. Once** with the original survey and once again with the "door to door" resurvey. We are continuing and will continue to resurvey the entire Island concentrating our efforts on known areas of high prevalence and on age groups expected to yield the highest results in seropositivity.

Although our case finding project has been rather extremely reduced in size it has continued to function effectively. I believe we may safely expect to test 50,000 persons this year with one team consisting of one and sometimes two nurses. However, as we complete testing in more heavily populated areas and move out into the smaller towns we find it increasingly difficult to maintain a high yield —either in terms of total specimens taken or total positivity.

We have been conducting a mass serologic survey in Puerto Rico for three and one-half years and the effect on the prevalence of syphilis is being demonstrated in the drop in cases reported. It becomes daily more difficult to find a new case of syphilis —as is graphically shown in this chart of morbidity reported by stage over the eight quarters of 1953 and 1954.

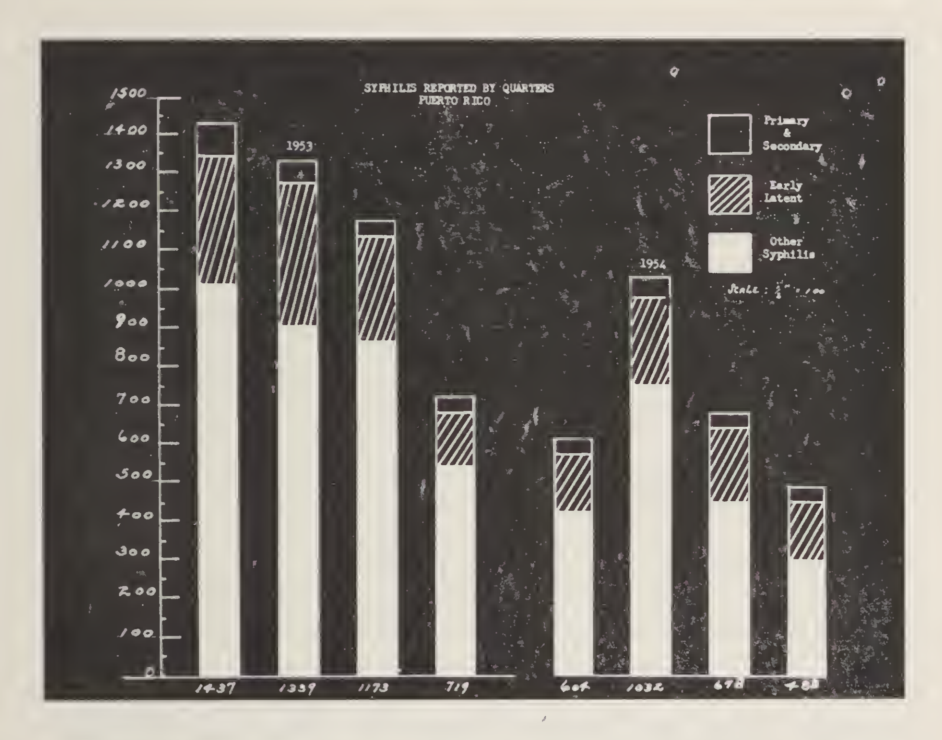
Over a period of five years the number of cases of infectious syphilis per 10,000 population has declined from 2.76 in 1950 to .83 in 1954.

Primary and Secondary Syphilis Cases Per 10,000 Population Puerto Rico — 1950-1954

Year	No. Cases	Rate Per 10,000
1950	608	2.76
1951	419	1.88
1952	384	1.71
1953	269	1.20
1954	186	.83

* Presented at the Venereal Disease Control Seminar, U.S. Region III, IV and VII of the Public H. Service, Miami Beach, Florida, Apr. 20-21, 1955.

** Director Division of Public Health and Acting Chief, Bureau of Venereal Disease Control, Department of Health of Puerto Rico.



However, we are not trying to lull ourselves into a feeling of false security — nor tell ourselves — or you — that syphilis is under control in our Island. Our little corner of the world, although small, is a busy crossroads where many people of many different countries meet. We have all the elements of an explosively dangerous situation as far as venereal diseases is concerned. We have a small population, relatively free from syphilis, but a population which is moving and changing. Our people are changing from an agricultural society to an industrial society. The industrial revolution — which is a thing of the past in most countries —is just now getting into full swing in Puerto Rico. Our people are moving from the country to the city and from the Island to the mainland. This is accompanied by the usual unrest found in such situations. Social mores are changing —family ties are breaking down — the status of women is also changing. These things, we know from experience, are dangerous factors in the venereal disease picture.

At the same time — more than ever — we have groups from the outside coming into constant contact with our people. One group is the tourists — with whom we must class the thousands of U. S. Marines and Sailors who visit our Island during the annual maneuvers. This group has frequent sexual contact with our population and if infection occurs it can easily be carried back

to the mainland. Unfortunately this group of Service men has ample opportunity to be exposed to venereal infection in other Caribbean islands before arrival in Puerto Rico, and in most of these other islands venereal diseases is totally uncontrolled.

The other group that comes into contact with our people has less opportunity for sexual exposure but perhaps more chance of spreading infection. I refer to the people of the other islands of the Caribbean, who do not enjoy the advantages of our public health control measures nor the blessings of our economic system and who wish, for one reason or another, to get into our country, to become citizens or work as temporary or migrant laborers. The contact of Puerto Ricans with these people is mostly through the Virgin Islands. As you perhaps do not know, a large portion of the population of the Virgin Islands, particularly St. Croix, is Puerto Rican. Into the Virgin Islands, particularly St. Croix, come large numbers of people from Tortola, Antigua, Barbados and many other islands of the British West Indies. As our "British cousins" they are almost as free to come and go as we Americans are and the very nature of the islands make it impossible to control their movement. They all come from areas where syphilis is highly prevalent and where control measures are non-existent.

Such a potentially explosive situation as I have described must be watched with the same eternal vigilance that is the inevitable price of freedom. A city does not close its fire stations and sell its fire trucks because it has not had fire for a year or two nor does the prudent home owner cancel his fire insurance policy because his house has never burned down.

We in Puerto Rico hope to continue a dual program designed to effect the reduction of venereal infection — at the same time guarding against the sudden outbreak of an epidemic. We will continue to have a mobile blood testing unit to work in areas of high prevalence — always ready at a moments notice to move to communities where new outbreaks of venereal disease threaten to appear. At the same time we will depend on our epidemiologic program to break chains of infection through contact interviewing and investigation.

A very effective part of our epidemiologic program is the prophylactic treatment of known promiscuous persons, not only those who have been named as contact but also those who are potential carriers of infection. The effectiveness of this sort of prophylactic safeguard has just been demonstrated during spring maneuvers of the Marines on our little island of Vieques. This tiny island, a few miles off the eastern end of Puerto Rico, is the place where annual Marine amphibious maneuvers are held. There is a native population of about seven or eight thousand persons

and a Marine population of perhaps 10,000 —but these Marines are not permanently stationed. They come in groups and stay for a few weeks and then go back to the mainland to be replaced by another group. Among the native population there are 20 or 25 women who extend unlimited hospitality to the Marines. This group of women is rather closely supervised by both the local health authorities and the local police authorities. New girls are not allowed to come in from the main island and the girls who form this group are required to cooperate with the health department by reporting to the clinic — where they receive large injections of Bicillin at regular intervals. This has been going on for several years now and to date we have not had one case of syphilis among the Marines that could be traced to a source on Vieques— or anywhere else in Puerto Rico for that matter. I may be mistaken and certainly this is a statement difficult to prove but to my knowledge no Marine, among the many thousand who have been here, has ever caught a case of syphilis in Puerto Rico. This year we have kept careful records and we know that out of the 10,000 Marines who have been in Vieques there have been only 15 cases of gonorrhea from exposures occurring on Vieques. A group of 19 girls who were regularly injected with Bicillin were questioned as to the number of contacts they had with Marine Corps personnel. They stated that as a group they had a total of 2,405 contacts from February 19 to April 1, an average of 126 per girl. These contacts resulted in 15 cases of gonorrhea and no syphilis or other V.D. This is a rate of one case per each 160 exposures. We think this is a rather good record and a rather good indication of the effectiveness of prophylactic treatment with long lasting penicillin compounds.

We feel sure that if we can increase the use of long lasting penicillin as a preventive measure to forestall the development of both syphylis and gonorrhea and continue our mass blood testing activities to lower the residual infection we can continue the dramatic drop in venereal disease rates in Puerto Rico.

PAGINA DEL PRESIDENTE

NUESTRA LUCHA



Durante los últimos meses hemos laborado intensamente con la cooperación de muchos compañeros para tratar de que no se desvirtuara el propósito del Proyecto del Senado 625, que era el de extender por dos años los privilegios de la Ley 383, pero "sólo con respecto a aquellos médicos que estuviesen cobijados por dicha Ley al expirar ésta el 30 de junio de 1955."

Argumentamos ante los dirigentes de la Comisión de Salud y Beneficencia de la Cámara de Representantes, que ya era tiempo de terminar con este sistema de importar médicos al país, que había demostrado ser muy deficiente, especialmente en cuanto a la calidad de éstos y que resultaba altamente perjudicial a la juventud puertorriqueña que en número mayor de 800 cursan estudios de medicina en nuestra Escuela de Medicina, en Estados Unidos y en el exterior.

La Legislatura, sin embargo, a petición de los alcaldes, eliminó la cláusula correspondiente, autorizando así a que se sigan importando médicos extranjeros hasta junio 30 de 1957, basándose en la falacia de que existe una "emergencia" y una gran "escasez" de médicos en nuestra Isla.

Muy poca consideración se le dió a los argumentos de peso presentados por nuestra Asociación fundamentados en estadísticas reales, y el proyecto fué aprobado y está actualmente ante la consideración del honorable Gobernador.

Es evidente que muchos alcaldes y algunos legisladores se preocupan más por las elecciones del año que viene que por la salud del pueblo y el porvenir de nuestra juventud.

Seguiremos luchando hasta lo último para proteger la salud de nuestro pueblo y los derechos de nuestros estudiantes de medicina.

* ✧ *

Hay otra nube mayor y más negra todavía en el horizonte para la clase médica y para todas las clases profesionales de Puerto Rico: el sustituto del Proyecto del Senado 511.

Dispone este proyecto la eliminación de todas las Juntas Examinadoras, traspasando sus poderes a los Secretarios del Gabinete con el propósito de "asegurarle al pueblo de Puerto Rico mayor protección a la vida, la salud y la prosperidad, y eliminar de una vez y para siempre los privilegios establecidos por grupos que en

el pasado han sido tan funestos para la buena marcha del pueblo", según se expresa en la 'Exposición de Motivos' del mismo.

Esta forma de mancillar a todos los prestigiosos y abnegados profesionales que han aceptado la encomienda héchale por el propio gobierno para servir en las Juntas Examinadoras de las distintas profesiones, a nombre de la justicia y la democracia, es indigna de una Comisión Legislativa y merece nuestro reproche y el de la propia Legislatura.

No dudamos que el sentir de los dentistas, farmacéuticos, enfermeras, ingenieros, arquitectos, agrónomos y otros profesionales incluídos en esta medida legislativa, ha de ser el mismo de la clase médica, que siente el ineludible deber de abogar ante la Legislatura y ante el poder ejecutivo por que no se cometa tamaña injusticia con los médicos que han formado parte de la Junta Examinadora, y sobre todo porque no se pretenda corregir irregularidades que haya podido cometer alguna junta, mediante acción tan antidemocrática y dictatorial como la recomendada por este proyecto.

Poner en manos de los secretarios del gabinete todos los deberes y poderes para dar y quitar licencias a los profesionales y artesanos sería un paso retrógrado en nuestro sistema democrático de gobierno y tendría un efecto diametralmente opuesto al que persiguen los auspiciadores del proyecto.

Accediendo a nuestras insistentes solicitudes, la Comisión que preside el Senador Heraclio Rivera Colón nos ha ofrecido celebrar próximamente vistas públicas en relación con dicho proyecto, de manera que podamos todos exponer nuestros puntos de vista y que tenga la oportunidad la honorable Legislatura de conocer el verdadero proceder de las distintas juntas en el cumplimiento de sus obligaciones.

Suplicamos a todos estar preparados para cuando hayan de celebrarse las vistas públicas en relación con esta medida legislativa.

* ✧ *

Un tercer proyecto que nos ha preocupado mucho ha sido el P. de la C. 1348, cuya finalidad es eliminar de la Ley Médica el requisito de ciudadanía. En nuestras entrevistas con los legisladores amigos, pudimos convencerlos de que esta medida no es práctica y se nos prometió que la misma sería retirada. Confiamos en que los propulsores de ésta cumplan su ofrecimiento.

NOTAS ACLARATORIAS

El doctor Egidio S. Colón Rivera, nos llama la atención hacia el hecho de que su trabajo sobre '*Pulseless Disease*' publicado en nuestra edición de febrero de 1955, (pág. 76) fué hecho en colaboración con los doctores Francisco Litchtenberg y Carlos Guzmán Acosta. Por haber usado nosotros la copia no revisada del artículo los nombres de los coautores no aparecieron en nuestro Boletín. Lamentamos la omisión.

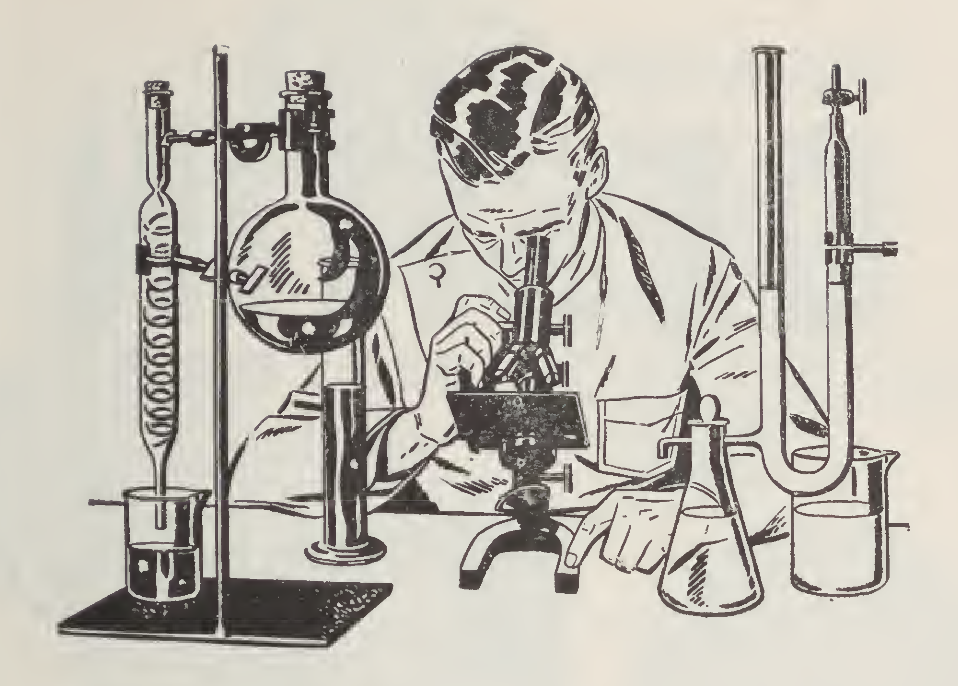
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En relación con el artículo 'Statistics about Physicians in Puerto Rico" por el doctor Oscar Costa-Mandry, publicado en la edición de marzo de 1955, pág. 113, se nos ha pedido hagamos las siguientes aclaraciones:

> En la página 117, entre el grupo de pediatras, aparece el nombre de la doctora Eloísa Muñoz Dones como si no tuviera el board americano de la especialidad. La doctora Muñoz Dones aprobó sus exámenes en octubre del 1954.
>
> En la página 118, entre el grupo de cirujanos generales aparece el doctor Francisco L. Raffucci con una nota al efecto de que ha hecho estudios postgraduados con beca del gobierno. Nos informa el propio interesado que él nunca ha disfrutado de tal privilegio.

Lamentamos sinceramente estos errores.





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Referencias: (1) Doyle, J. C.: Urol. & Cutan. Rev. *55:* 618, 1951; (2) Weinstein, B. B., y Weinstein, D.: Mississippi Doctor *29:* 117, 1951; (3) Schwartz, J.: Am. J. Obst. & Gynec. *63:* 579, 1952; (4) Schwartz, J., y Nardiello, V.: Am. J. Obst. & Gynec. *65:* 1069, 1953.

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DE LA

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BOLETIN

DE LA ASOCIACION MEDICA DE PUERTO RICO

VOL. 47 MAYO, 1955 No. 5

PORTAL HYPERTENSION

TRANSPOSITION OF THE OMENTUM INTO THE MEDIASTINAL SPACE AND LEFT THORACIC CAVITY, WITH LIGATION OF ESOPHAGEAL VARICES, SECTION OF VARICES AND ESOPHAGUS, AND ESOPHAGO-GASTROSTOMY; REPORT OF FOUR CASES.

*LUIS A. PASSALACQUA, A.B., M.D., F.A.C.S.**

The accepted opinion today of the majority of the surgeons is that the only rational treatment of Portal Hypertension is the establishment of a shunt, either between the splenic and the renal veins, with the accompanying splenectomy, or between the portal veins and the inferior vena cava.

The literature on this subject is very extensive and it is entirely outside the scope of this paper to discuss any of it.

On the other hand, the literature does not present one single instant in which the omentum is brought up into the mediastinal space and the pleural cavity with the intention of facilitating the portal outflow by inviting the formation of a collateral circulation between the portal system and the systemic circulation by way of **the omentum, with the vascular structures of the mediastinum and pleural cavity.**

This type of operation was first performed by me in the Veterans Administration, San Patricio Hospital, San Juan, Puerto Rico, on January 25, 1949, and until January 1951, four such operations were performed. One of these patients had a liver death six days after the operation.

Since over four years have already elapsed since the last operation, it seems to me that it would be of some general interest to report the cases that survived the operation.

I would like to mention that in 1947, D.B. Phemister and E.M. Humphreys described one case in which the lower portion of the esophagus and the upper portion of the stomach were resected to

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* Chief, Surgical Service, V. A. Hospital San Patricio, San Juan, P. R.

NOTE: The statements and conclusions published by the author are the result of his own study and do not necessarily reflect the opinion or policy of the Veterans Administration.

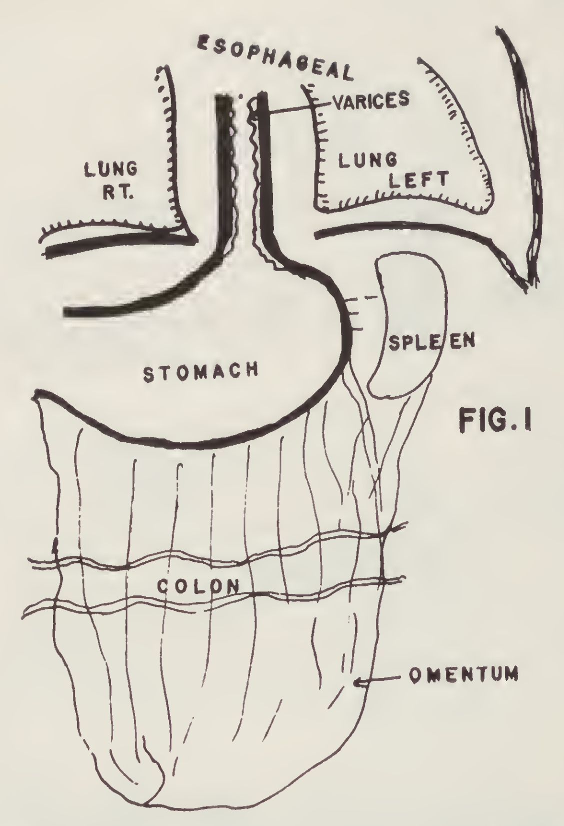


Figure 1. The purpose of this operation is to invite the formation of a collateral circulation between the omentum and the vessels of the thoracic cavity. For that purpose the omentum is brought up into the mediastinum and the left pleural cavity where it is spread over the lower portion of the lung, the esophagus, over the diaphragm and into the mediastinal structures. The varices are attacked directly by ligation and section, followed by an esophago-gastrostomy.

control bleeding from the esophageal varices. These authors also reported a case of total gastrectomy for the same condition. They also removed the spleen. Several successful cases of partial esophago-gastrectomy were also reported by H. D. Adams in 1950, P. W. Shafer and C. F. Kittle also in 1950 and by Carl Axel Ekman and Phillip Sandblom in 1954.

Although the stomach was not resected in any of my cases, the esophagus was cut near its gastric end, and the mass of dilated veins was ligated near the stomach.

The spleen was not removed except in one case with hypersplenism, but an esophago-gastrostomy was made connecting the esophagus to the fundus of the stomach and a portion of the stomach was brought into the pleural cavity. (Fig. 6)

It was noted that after section of the esophagus, ligation of

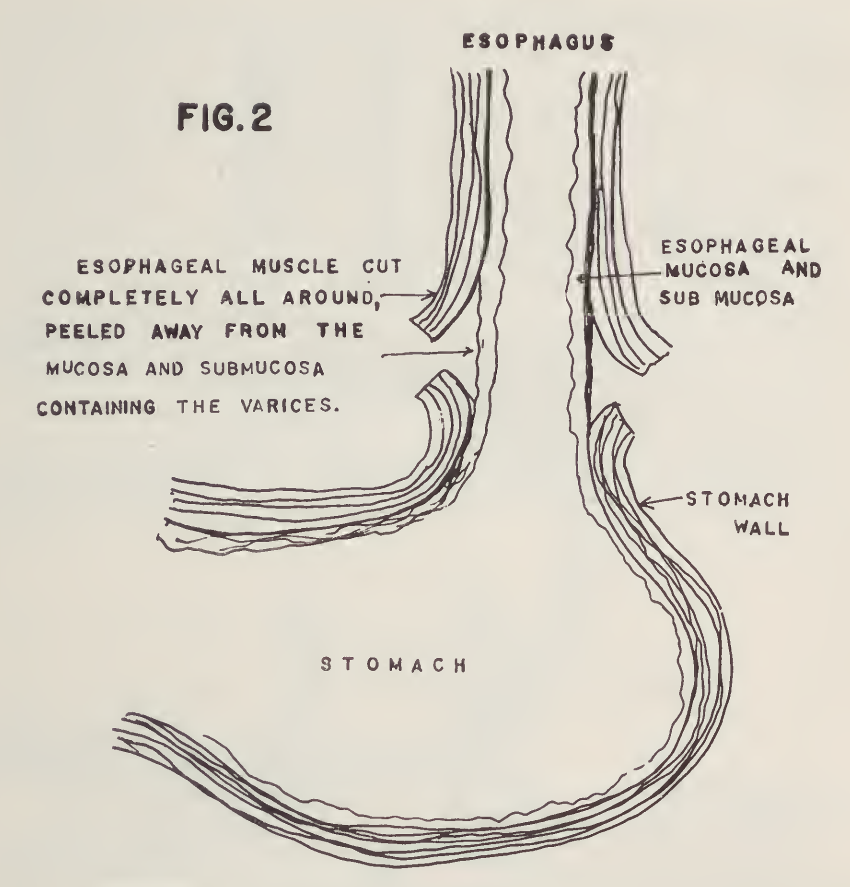


Fig. 2. The esophageal muscle is cut completely around the esophagus, and is dissected by blunt dissection away from the submucosa and the mucosa, separating it from the varices of the submucosa.

the ends of the esophageal varices in the cephalad end of the esophagus was not necessary, since they immediately collapsed and the esophago-gastrostomy could be performed in the usual way. (Fig. 3)

The position of the patient on the operating table was the face down position described by Overholt for pulmonary resection. I have been using the face down position for esophagectomy since 1948 and find it to be very satisfactory and convenient.

Case No. 1 — A 60 year old veteran of the first World War was admitted to the VA Hospital San Patricio, October 27, 1948 because of severe hematemesis. He had Schistosoma mansoni in the stools. The past history reveals that he had been hospitalized elsewhere for moderately advanced tuberculosis in 1946 and stayed as an inmate in a TB Sanatorium until his transfer to the VA Hospital San Patricio. During his stay in the TB Sanatorium he

received Fuadin for Schistosoma mansoni and also marpharsen for syphilis. In 1947 he had a profuse hematemesis. Roentgenologic study of the stomach demonstrated a duodenal ulcer. No barium swallow was made at that time to demonstrate esophageal varices. In 1948 he had a second severe hematemesis and transfer was made from the sanatorium to our hospital.

On admission the spleen was found filling the left side of the abdomen to the umbilicus and below the left iliac crest. (Fig. 7). The liver was small. Ascitis was present. Hanger test 4+. Icteric index +12. Total blood proteins 7.2 gms. (Albumen 2.7 gms., globulin 4.5 gm.) Red blood cells 2.8 millions. X-ray of the chest showed "chronic tuberculosis, moderately advanced, involving both upper lobes". Repeated sputum examinations were negative. G. I. series showed a large niche in the prepyloric region. Barium swallow demonstrated large esophageal varices. Following preparation with marked improvement, the operation was performed January 25, 1949 as follows:

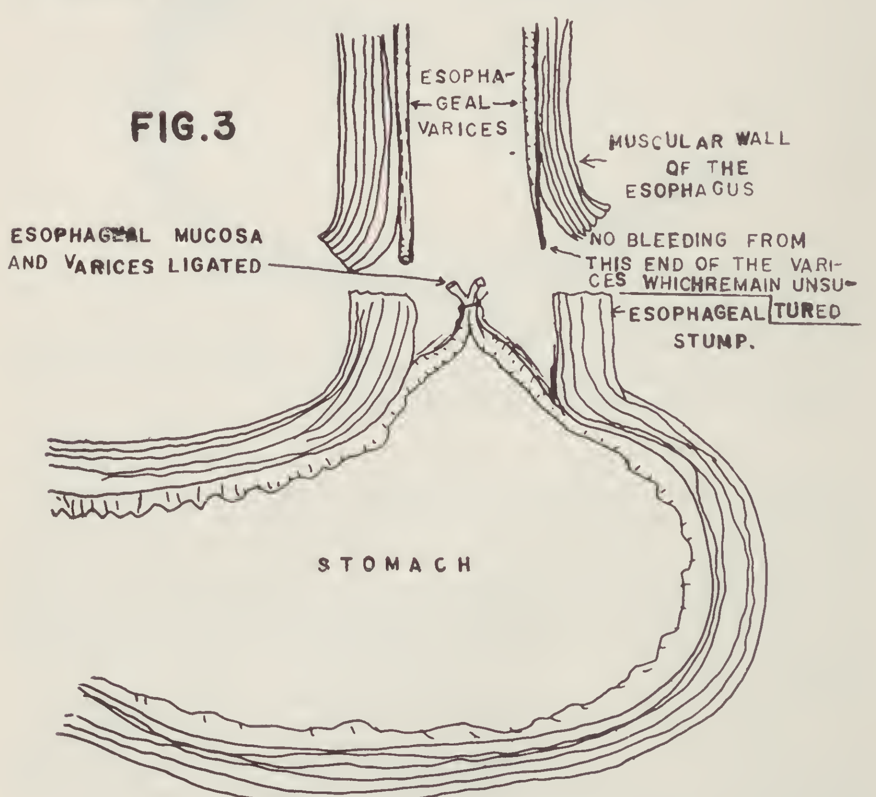


Fig. 3. The esophageal varices have been separated from the muscle layer, the mucosa and submucosa with the varices have been tied with silk No. 1, close to the stomach, and cut across. The proximal end is left untied, as it immediately collapses, and then there is no bleeding or evidence of suction.

FIG. 4

ESOPHAGUS

VARICES DO NOT BLEED AFTER SECTION MAKING LIGATION INNECESSARY AT THE ESOPHAGEAL END.

DIAPHRAGM

INVERSION OF THE LIGATED ESOPHAGEAL STUMP AND SUTURING OF WALL OF STOMACH OVER IT.

STOMACH

Fig. 4. The esophageal stump is inverted into the stomach and a second row of sutures is placed over it completing the closure of the stomach opening.

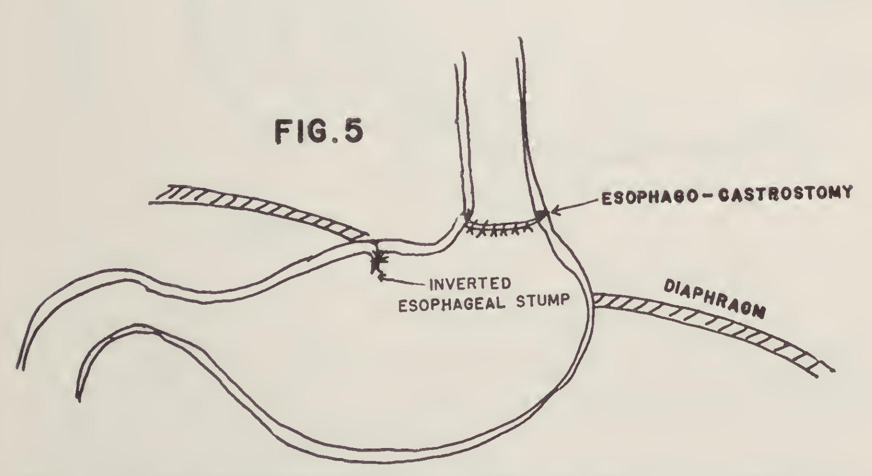


Fig. 5. A new esophago-anastomosis is made connecting the esophagus to the fundus of the stomach.

With the patient in the face down position the left side of the pleural cavity was entered thru the bed of the 7th rib. The diaphragm was opened at the central tendon and the esophagus and stomach mobilized without disturbing the circulation of the stomach. The fundus of the stomach was brought above the diaphragm together with the omentum. The muscular wall of the esophagus was cut near the gastric end with a circular incision, (Fig. 2), and the muscle coat pushed upwards and downwards. The mucosa of the esophagus and the large varices were clearly demonstrated and tied with #1 silk. (Fig. 5). The omentum was brought over, (Fig. 6), fixed by several catgut sutures to the lower surface of the lung, fan shape manner, around esophagus and to the

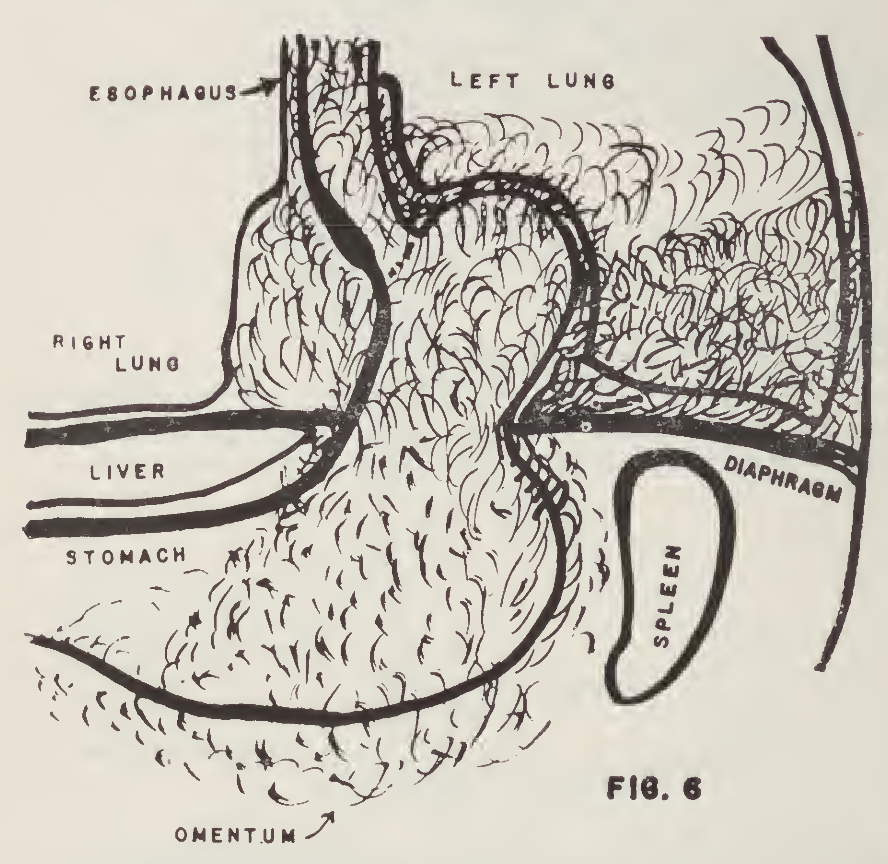


Fig. 6. The fundus of the stomach is seen in the thoracic cavity, following esophago-gastrostomy. The omentum is also represented filling the mediastinal space, surrounding the esophagus and the lower portion of the lung completely. A thin layer of talcum powder was smeared between the contact surfaces of the omentum, lung, pleura and diaphragm to stimulate granulation. The autopsy in a patient who died 9 months after operation showing advanced tuberculosis with extensive cavity formation, demonstrated the presence of markedly dilated veins and adhesions between omentum and the intrathoracic structures.

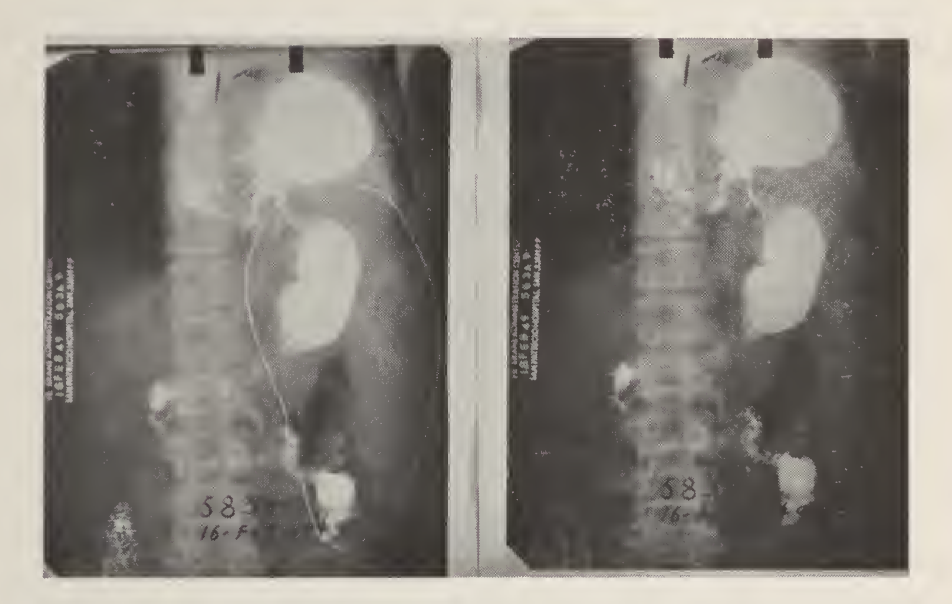


Fig. 7. - Case I. Roentgenograms demonstrating the size of the spleen before the operation. At autopsy the spleen was found greatly reduced in size, weighing 400 gms.

loose tissues in the mediastinum. A thin coat of talcum powder was spread on the surface of the omentum, lung and diaphragm to stimulate the formation of adhesions between these structures. The patient went home in good condition. Eight months after discharge he was called for follow-up studies. He was admitted to the hospital in an advanced stage of malnutrition and tuberculosis, with peripheral edema and some evidence of ascitis. He claimed his home diet was very poor. Did not have hematemesis since operation. He died November 9, 1949. Autopsy demonstrated the following significant findings: (1) Lungs: extensive tuberculous cavitations of both lungs. (2) Definite evidence of collateral circulation between omentum and the structures in the mediastinum, surface of the lung, pleural wall, diaphragm, and between the contact surfaces of the intrathoracic portion of the stomach and the diaphragm, the left lung and the pleuritic wall. (3) Thrombosis of the mesenteric vein. (4) Small amount of ascitis. (5) Greatly diminished size of the spleen, as compared to the size before the operation, (Fig. 7), weight 400 gms. (6) Small cirrhotic liver. (7) No evidence of esophageal varices.

Of apparent importance from the point of view of the operation is the formation of a collateral circulation, the absence of ascitis until the terminal stage of malnutrition, the marked diminution in the size of the spleen.

Case No. 2 — A 28 year old veteran with a history of in-

fectious hepatitis and jaundice in 1948 while in the Army. No history or evidence of schistosomiasis. Previous to his admission December 15, 1949 to the VA Hospital San Patricio, he suffered two severe hemorrhages (hematemesis). The existence of cirrhosis of the liver, splenomegaly and esophageal varices was confirmed. On July 11, 1950 the operation was performed as described in Case No. 1.

The patient had pan-hypersplenism, and the spleen was removed January 16, 1952. The patient today is working, is strong and feels well. Hematemesis had not recurred. No ascitis.

Case No. 3 — Veteran, aged 40, who was treated with Fuadin because of Schistosoma mansoni. The liver and spleen were easily palpable and the X-rays showed extremely large esophageal varices. The patient was rather obese and no evidence of ascitis was detectable. He did not have bleeding episodes, although his blood count was low. The bromosulfalein showed 38% retention in 45 minutes and a Hanger of 3+ in 48 hours. No evidence of hypersplenism. The operation was performed March 12, 1951 as described in Case No. 1: The esophagus was cut across, the varices ligated, the esophagus anastomosed to the fundus of the stomach, the fundus of the stomach and the omentum brought up into the thoracic cavity and fixed in the mediastinum and base of the lung. Recovery was uneventful and his condition today is very good. He reports "good health". The liver is still palpable and the spleen is also palpable. No liver function tests have been done lately but the patient reports that he is working well and in very good condition. No hematemesis. No melena. No ascitis.

CONCLUSIONS

1. These three cases represent a very small series and no conclusions are attempted. They are presented for what they are worth. Besides, very little time has elapsed between the operation, four years nine months in the second case, four years one month in the third case. Outside of some esophageal regurgitation, these two men are in very good condition today and to all appearance, the varices have disappeared.

2. Perhaps this operation can be simplified by incising the esophagus, ligating the varices, as recommended by Crile, and, without mobilizing the stomach, bringing the omentum into the thoracic cavity. In this way the esophagus is not cut, there is no

need of doing an esophago-gastrostomy and the sphincter in the cardia of the stomach is preserved.

3. The first case demonstrated at autopsy the formation of collateral circulation, the marked decrease in size of the spleen and the disappearance of ascitis, until the terminal stage, when extensive tuberculous cavitations, mesenteric thrombosis and severe malnutrition were present.

PREVENTIVE MEDICINE FROM THE PHYSIATRIC POINT OF VIEW*

*HERMAN J. FLAX, M.D., M.Sc. (Phys. Med.)***

A previous paper,[1] delivered several years ago during a similar assembly, discussed the physiatric methods of preventing the complications of complete bed rest. The following presentation will emphasize the "disuse atrophy of soft tissues" and suggest some simple means of preventing this condition in ambulatory patients. Usually, by the time this complication is recognized, it is more disabling and requires more treatment than the original diagnosis. This constitutes an added expense and a loss of earning power for the laborer, a shortage of hospital beds and a scarcity of treatment opportunities for other patients.

The selection of some characteristic case histories will stress the importance of the "disuse atrophies" as the main reason for continued disability in postural and post-traumatic deformities.

POSTURAL DEFORMITIES

Strange as it may seem, postural pain knows no age limit. The following three patients are typical examples.

(1) "Growing Pains"

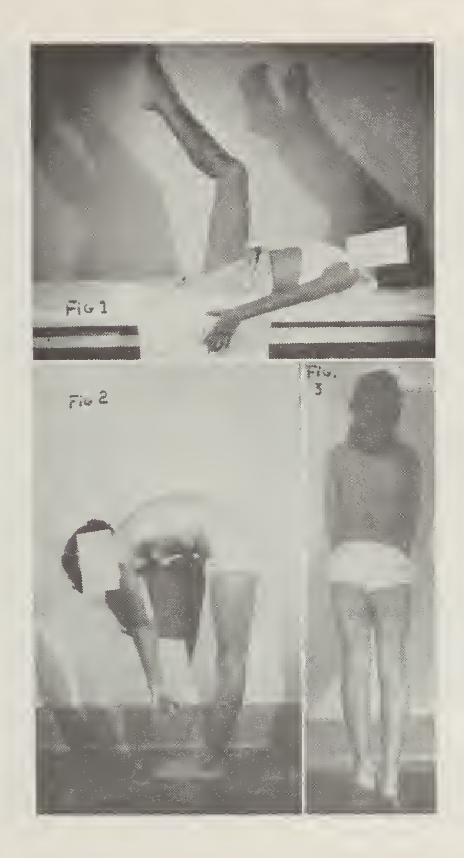
A young girl of eleven years was seen because of severe night cramps in the lower extremity muscles of several months' duration. The parents paid little attention to this complaint, because the child was always healthy and did not show any constitutional signs. One night, however, the girl cried so much that the parents became alarmed and sought medical advice. Questioning revealed the fact that suddenly the child began to grow more rapidly, and she was taking dancing lessons. There was no past history of infectious diseases or trauma.

The most interesting discovery was a marked contracture of the hamstrings and a lesser shortening of the calf muscles bilaterally. (Figures 1, 2, 3). There was no muscle weakness and no limitation in range of joint motion. The calf muscles had numerous tender, spastic muscle areas, pressure on which reproduced in part the night pains.

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* Presented at the 51st Annual Meeting of the Puerto Rico Medical Association, Dec. 10, 1954.

** Chief Physical Medicine and Rehabilitation San Patricio VA Hospital.



This condition is called "growing pains", and once rheumatic disease is ruled out, usually, nothing more is done. This results in a permanent shortening of the hamstring muscles and occasionally the calf muscle, continued pain from muscle spasms and a curtailment of the normal child's play activities with probable emotional conflict. Finally, since the hamstrings originate from the ischial tuberosities, they will pull the pelvis backward and downward with a resulting strain on the lumbosacral articulation and surroundig soft tissues giving rise to chronic low-back pain and poor posture. (Figure 4).

The proper treatment for this patient is complete rest for a few days with local heat and light sedative massage to relieve the muscle spasm. Afterwards, a series of exercises to stretch the hamstrings and calf muscles is prescribed. Care must be taken that this program gradually stretches the muscles; otherwise, multiple minute muscle and connective tissue tears will cause more fibrosis and more shortening. When the muscles are stretched to their normal length, calisthenic exercises and stretching a.e continued daily over a prolonged period, preferably forever, to prevent a recurrence of these contractures.



(2) **"Low-back Pain"**

The second patient is a civil engineer, 39 years, referred by a urologist, who complained of a low-back pain of many years' duration. This pain was localized in the lumbosacral region, did not radiate, was more pronounced upon arising in the morning and did not improve with activity. The pain was a dull ache, never sharp and always present in the middle of the lumbosacral joint. Lately, the patient had become so conscious of this pain that it interfered with his work. The patient mentioned that he was an athlete during his university days but over the past decade had exercised very little. He had gained a great deal of weight lately.

The patient was of stocky build with no muscular weakness and no limitation of joint motion. There was a marked contracture of the hamstrings bilaterally. The legs extended only to 45 degrees with the hips flexed, when normally, they should extend to a right angle. There was pain on pressure over the lumbosacral region. Several tender, spastic muscle masses were palpated in the lumbo-paravertebral and gluteal regions. The patient lacked 13 inches to touch the floor with the knees extended. Neurological examination was normal. (Figures 5, 6).

This is a very common ailment, classified in that vague hodgepodge of the "low-back syndrome". It is a definite clinical entity



Figs. 5 & 6

diagnosed as a "Postural Defect", caused by disuse atrophy and subsequent contracture of the hamstring muscles. These muscles, as was mentioned previously, originate from the ischial tuberosities, and when they are stretched, they pull the pelvis downward and backward. This tug-of-war between the strongest muscles in the body and the lumbosacral joint traumatizes this region, causing lumboparavertebral muscle spasm and pain. An unusual strain, such as suddenly slamming on the brakes of the automobile, the back-bending of the week-end gardener or lifting an object (it need not be heavy!) with the knees straight, will precipitate a serious trauma with laceration of the tissues around the lumbosacral and sacroiliac joints and a sprain of the lumboparavertebral muscles. This gives rise to a severe low-back pain and in some patients prolonged disability. Mention only can be made of spastic flat feet and degenerative arthritis of the knee joints, secondary to this postural defect.

Treatment of this condition is aimed primarily at the prevention of repeated traumata to the soft tissues of the low-back

region by relieving the constant pull of the hamstring muscles and establishing a program to stretch the contracted muscles gradually. In the acute stage, bed rest on a firm, non-ceding mattress for several days is the best treatment. As soon as the patient can get out of bed, short-wave diathermy for thirty minutes to the lumbosacral and gluteal regions is advocated followed by deep sedative and friction massage to the tender, spastic muscle areas. As soon as the spasm is released, the massage is discontinued, and manipulation of the low-back is done. This is followed by gentle stretching of the hamstrings. A program of progressive and resistive exercises is prescribed as soon as the acute pain has disappeared. Usually, the dull ache is relieved in three to four weeks, at which time, the patient is discharged from office treatment. The exercises and stretching are continued at home in increasing amounts over several months or until the hamstrings have attained normal length.

In many instances, it will be impossible to achieve this goal. This is especially true of the older patient. It is for these persons that the following precautions are most important. Simple measures, as sleeping on a firm mattress, using posture chairs that support the curves of the back and permit the feet to reach the floor easily without any pressure against the popliteal space and moving the automobile seat a few inches forward to relieve the tension of the hamstrings, may be all that is required to prevent low-back pain. Insofar as possible, the patient should sleep on top of the mattress, sit on top of the seat and not be engulfed or deceived by the softness of the support. Although difficult for many people, their sleeping posture should be changed, so that they sleep on their sides with the knees and thighs slightly flexed. If they must sleep on their backs, a firm pillow should be placed under the knees. In addition, the patient is taught to lift objects from a squatting position by extending the thigh muscles and not the back. A program of progressive resistive exercises to strengthen the gluteal, back and abdominal muscles is prescribed. At all times, the patient is made conscious of his body posture in all positions.

(3) The Executive Back".

The third problem of faulty posture implicates the aches and pains in the intrascapular region that radiate up into the neck and across the shoulder. This was the chief complaint of a physician, 34 years, who came to me in desperation. The diagnosis was relatively easy to make in this patient, because he had ruled out everything else through consultations with several colleagues.

Palpation revealed an exquisitely tender spot along the vertebral border of the left scapula and especially over the medial angle. Pressure on this area immediately provoked the sharp, shooting pain that radiated up into the left occipital region and also across the left suprascapular area. This syndrome is a common occurrence in the executive or white-collar working class.

The exact reason for the formation of this "trigger" area is not known. About as much is known about the formation of the "fibrotic" nodules easily palpated in the trapezii and other shoulder muscles in these individuals. Whether or not this is a muscle fatigue pattern, brought about by poor posture and resulting disuse atrophy of the muscles connecting the upper extremities to the vertebral column, is speculative.

Another explanation might well be this. In a sedentary occupation that does not require much lifting or abduction of the upper extremities, a disuse atrophy of these muscles occurs. Suddenly, for some reason, the patient is called upon to abduct his shoulder suddenly and forcefully as in carrying a suitcase, lifting weights for the first few times, working in the garden or steering an automobile. This results in minute tears in the muscles and their tendinous attachments. These heal with a scar that is less elastic than the original muscle, peritendinous or tendinous tissue. These areas are more subject to repeated tears. As a result, a focus of irritation is established. This stimulates a reflex pattern of pain, spasm, more pain, more spasm, going around in a viscious circle. This reflex can be broken only when the focus of irritation or "trigger" area is eliminated.

The best method of accomplishing this, in my opinion, is to infiltrate the area with a few cubic centimeters of one per cent Procaine Hydrochloride Solution, and as soon as the anesthesia takes effect to forcefully massage the region with the thumbs. The fibrotic nodules in the muscles also should be infiltrated with local anesthesia, as they are quite tender to touch. Usually, three or four treatments of local anesthesia and friction massage are required at intervals of 48 to 72 hours before the patient is able to tolerate the massage alone. Any other spastic sites in the scapular and cervical paravertebral muscles are also massaged until the spasm is released.

Next, the scapulae are manipulated to stretch their attachments. The head, neck and shoulder joints are carried passively through their complete range of motion. Lastly, a program of progressive resistive exercises for the scapular and shoulder muscles, head traction with a Sayre sling and posture exercises is continued daily at home for several months.

DISUSE ATROPHY AND CONTRACTURE OF SOFT TISSUES FOLLOWING TRAUMA

Perhaps the most serious complication following trauma is the disuse atrophy and contracture of soft tissues, either as a result of direct injury and scarring or secondary to immobilization in a poorly functional position. In many patients this deformity is the sole cause of disability; the original lesion having healed without any incapacity.

Three patients will be presented with typical deformities of disuse atrophy of soft tissues. In each instance, they recovered from the primary trauma. Treatment was directed to improve a complication that could have been prevented with early motion of the uninvolved joints and exercises.

(1) Injury to Knee.

On January 11, 1954, this patient received a slight blow to the right knee which became painful and swollen. He was seen immediately by a physician, who took roentgenograms and prescribed rest and heat. Within a week, the swelling had disappeared, but the patient continued to complain of pain. Local heat, using an infra-red lamp, was prescribed in a local dispensary for an additional two weeks. The patient was discharged and told to return to work. Nine months after the accident the patient was seen in consultation, because of pain, weakness and instability of the right knee. He was unable to work as a chauffer.

Moderate muscle atrophy of the right thigh was found (Figure 7), and there was tenderness to pressure over the medial condyle of the tibia. The range of motion was normal, and no lateral instability nor pain on extension was elicited. There was no synovial effusion. Roentgenograms were negative for any intra-or extra-articular pathology. All the symptoms were secondary to disuse atrophy of the quadriceps muscle following contusion to the right knee.

Quadriceps muscle atrophy follows rapidly any injury that restricts motion of the knee. My personal experience has shown as much as one-half inch atrophy of the thigh muscles in as little as 72 hours after the trauma. Unless the muscle is forced to regain its normal strength through progressive resistive exercises, this weakness will persist almost indefinitely.

In the early post-traumatic stage, even if the knee is immobilized in a cast, repeated quadriceps-setting and straight-leg raising exercises will prevent rapid muscle wasting. The patient will be made aware of the quadriceps muscle action and will learn its value in stabilizing the knee. As quickly as possible, a program

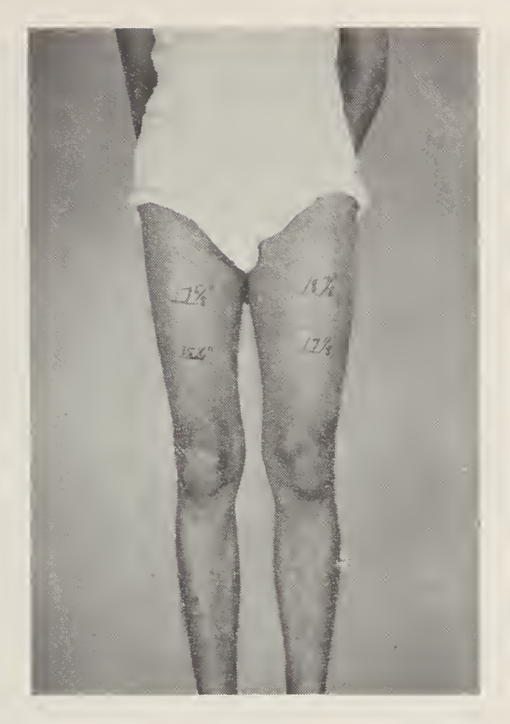


Fig. 7

of progressive resistive exercises is prescribed, using the technique described by DeLorme[2] or Zinovieff[3] every day. A practical stopping point for these exercises is to raise a weight equivalent to the amount elevated by the uninjured side. At this point, if there is still measurable muscle atrophy, all exercises are continued until both thighs are equal in size. The only way to insure a stable knee is to guarantee a strong quadriceps muscle.

(2) Limitation of the Shoulder Movements Following Forearm Fracture.

On December 6, 1953, this 42 year old man fractured the middle third of both left radius and ulna. There was malunion of the fragments, and on January 14, 1954, he was referred for an open reduction. This was not thought necessary at this time. A new cast was applied, and the patient was sent home. Because of non-union, on March 31, 1954, an intra-medullary nailing with bone grafts of both radius and ulna was done.

On April 12, 1954, after 18 weeks of immobilization and 2 weeks after surgery, the patient was sent to Physical Medicine and Rehabilitation Service for the first time. The fingers, hand and forearm were rigid. There was no active shoulder movements

above 90 degrees of abduction, and external rotation was zero. At his own request, the patient was discharged three days later, but he was instructed in a home program of pulley and grasping exercises for the shoulder and hand.

The patient returned on July 14, 1954 and was hospitalized on the Physical Medicine Rehabilitation Service for definitive treatment. Upon discharge on October 22, 1954, after receiving maximum benefit of treatment, the range of motion of the left upper extremity joints had progressed to the stage illustrated in Figures 8, 9. The fingers touch the palm; dorsiflexion of the wrist is 10 degrees, pronation is 60 degrees and supination is lost; the elbow moves from 45 to 90 degrees; and, the shoulder abducts to 80 degrees, flexes to 100 degrees and moves from 15 degrees of internal to 25 degrees of external rotation. It was felt, however, that the patient would continue to improve with active movements around the house and at work on the farm. A program of mobilization that really began 22 weeks following injury and 15 weeks after surgery can hardly hope to accomplish much more that the results shown by this patient.

It is well to point out that the limitation of interphalangeal and shoulder joint motion could have been prevented with early active and especially passive motion. This hastens the absorption of edema fluid, which glues the soft tissues around the joints and in muscles, delays disuse atrophy and prevents contractures of soft tissues.



(3) Permanent Deformities Secondary to Soft-tissue Contractures.

This 29 year old man was struck on the left forearm on June 18, 1954. Three surgical procedures were necessary during the first month to correct an oblique fracture across the proximal third of the ulna and an anterior dislocation of the head of the left radius. He was still in a cast from hand to upper arm on October 29, 1954, when he came on his own accord for physical therapy.

The deformities of the left upper extremity can be seen in Figures 10, 11. All the fingers and thumb are in full flexion with loss of extension and opposition. The wrist is in complete palmar flexion with loss of dorsiflexion, pronation and supination. The elbow moves from 45 degrees extension to 80? The shoulder abducts to 60 degrees, is held in internal rotation of 30 degrees with complete loss of external rotation.

The appearance of this hand simulated a combined median and ulnar nerve palsy, but electrical stimulation showed response of all muscles to faradic and galvanic currents, and there was no sensory deficit. The picture presented is the result of an ischemic contracture of all the muscles below the elbow and a pericapsular fibrosis with disuse atrophy of the shoulder muscles.

Treatment of this condition is prevention. Once normal muscle fibers are replaced with fibrous tissue, very little can be done to

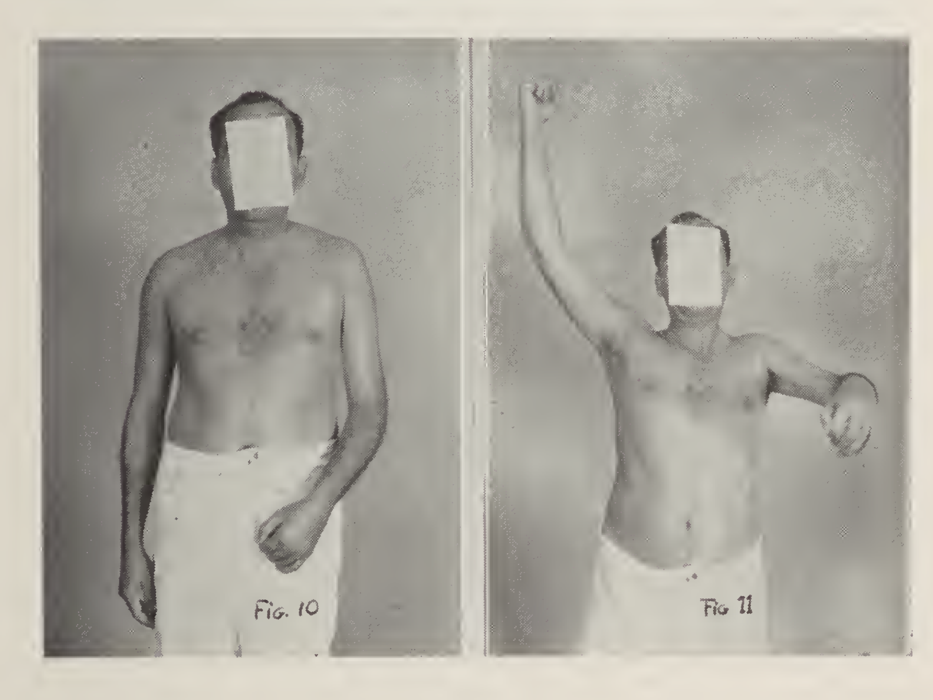


Fig. 10 Fig 11

restore the physiological properties of muscle. In the early stages, the use of mechanical devices, such as strong traction splints, continuous passive stretching and active movements of the joints may prevent the severe contracture deformities. Practically nothing can be done with physiatric modalities 19 weeks after immobilization. Nevertheless, treatment was ordered, and six weeks of manipulation and traction splinting proved ineffective. The patient was referred to the orthopedic surgeon for the possibility of specialized reconstructive procedures that may improve the function of the hand.

SUMMARY

Disuse atrophy and contracture of soft tissues, secondary to postural deformities and trauma, can be prevented by the early and continuous use of physiatric methods. In postural deformities the antigravity muscles, notably the hamstrings and the muscles connecting the upper extremities to the vertebral column, are involved. The hamstrings are contracted, and when stretched, they pull the pelvis back and downward, causing low-back pain. Careful palpation of the scapular, upper back and neck muscles will uncover multiple small, tender, spastic areas, which are secondary to muscle fatigue and are responsible for high-back pain. These pains can be relieved by rest, massage, stretching, progressive resistive exercises and proper body posture.

To prevent disability following an injury, the affected joints should be immobilized only when absolutely necessary and in the best functional position. The other joints should be carried through full range of motion several times daily beginning the day after the accident. As soon as possible, progressive resistive exercises should be prescribed to restore muscle strength of the injured and atrophied muscles.

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BRUCELLOSIS

PRESENTATION OF THREE CASES

*JENARO HADDOCK-SUÁREZ, M.D.**

Isolation of the Brucella organisms constitutes the only incontestable way of establishing a definite diagnosis of Brucellosis. Ruiz Castañeda[1] in 1947 described a method which has increased considerably the chances of obtaining positive blood cultures in human Brucellosis. In this method Trypticase Soy agar slants are prepared in various bottles where they are allowed to solidify after proper sterilization. A small amount of trypticase soy broth containing sodium citrate is added aseptically to each bottle and the specimen of blood is inoculated into the broth. Incubation is at 37ºC in an atmosphere of 10% carbon dioxide. The bottles are then tipped on their sides every 48 hours so that the agar slants may be bathed and reinoculated by the liquid portion. The organisms in the broth will thus grow more easily on the solid agar. In positive cultures growth is generally observed after the sixth day of incubation. The cultures are discarded as negative after 20 days of incubation.

By the use of this method we have been able to isolate Brucella abortus from the blood of three patients which were admitted to San Patricio Hospital with fever of unknown origin during the course of the last two years. Two cases were classified as acute Brucellosis, that is, an illness which has endured three months or less; one case as subacute Brucellosis referring to an illness whose symptoms have lasted from three to twelve months. These three cases are the subject of this report.

Case No. 1: is the case of a 69 year-old unemployed WWI veteran from Rincón who was admitted to San Patricio Hospital on February 1953 complaining of generalized pruritus and recurrent bouts of fever. The patient enjoyed good health until the latter part of October 1952, when he developed daily chills and fever and generalized pruritus. He received Penicillin and Gantrisin; the fever subsided and then he was well for about two months. He then again developed chills and fever. He was hospitalized at a VA contract hospital where he had daily spikes in temperature for one week. The patient received 14 gm. of Chloromycetin during the two weeks stay in the contract hospital. The agglutination titer for Brucella was 1:50 at that institution. He was then transferred to San Patricio Hospital for further studies. He admitted drinking raw milk obtained from cows he owned. He

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also admitted drinking considerable amounts of liquor in the last two years.

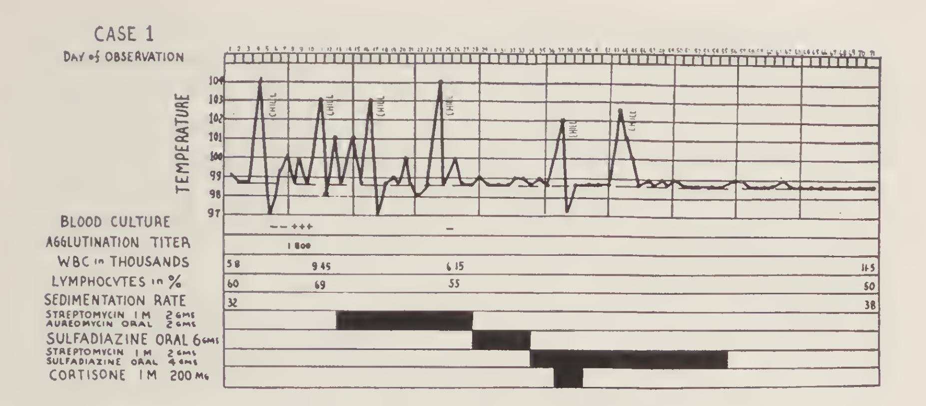
The positive physical findings on admission were: (1) A rough grade II aortic systolic murmur transmitted to the neck vessels. (2) An enlarged liver, the lower border of which extended three finger-breadths below the right costal margin. The liver edge was firm but not tender. (3) Splenomegaly — the tip of the spleen being felt one finger-breadth below the left costal margin.

The laboratory work revealed a white cell count of 5,800. The Schilling differential revealed 31% PMN, 60% lymphocytes, and 9% eosinophiles. The corrected sedimentation rate 30 mm. in an hour. The hemoglobin was 11.3 gm. The urinalysis was normal. The Brucella agglutination titer was 1:800. Blood cultures obtained on the 8th, 9th and 10th hospital days were positive for Brucella abortus. Liver function studies revealed: BSP retention of 8% in 45 min.; a 4+ Hanger test in 48 hours. Thymol turbidity 8.5 units, cholesterol esters 75%. Total serum proteins 8.3. A.G. Ratio 4.3/4.0. Needle biopsy of the liver showed edema of portal spaces which were infiltrated with polymorphonuclear leucocytes and lymphoid cells. There were a few focal areas in the liver parenchyma exhibiting evidence of degeneration associated with an infiltrate of polymorphonuclear leucocytes and few lymphoid cells.

X ray of the chest was negative. Intravenous pyelogram was normal. Barium swallow was negative for esophageal varices. X-ray of the lumbosacral spine revealed hypertrophic arthritis. X-ray of the sacroiliac joints was negative.

Therapy was started on the 14th hospital day with intramuscular Streptomycin 2 gm. daily and Aureomycin by mouth 500 mg. four times a day. These two antibiotics were given for two weeks during which the patient showed little improvement. He was anoretic and complained bitterly of low back pain. The patient was then given a short course of oral sulfadiazine followed by combined Streptomycin and Sulfadiazine therapy for three weeks. During the 6th hospital week he was given 3 days of intramuscular Cortisone because of severe low back pain and toxicity. A remarkable improvement followed, however the pain rapidly recurred on discontinuation of the cortisone. The patient became afebrile on the 44th hospital day and remained so for the rest of the period of hospitalization. The patient was seen by the consulting dermatologist in reference to the generalized pruritus. It was his impression that this had no relation to the rest of the clinical picture.

He was then seen one and a half years after discharge. There had been no recurrence of the chills and fever, no myalgias or arthralgias, no recurrence of the pruritic eruption. Physical exami-



nation revealed an enlarged liver and an enlarged spleen. The blood culture was negative; the brucella agglutination titer 1:100, the white cell count 5,500 with 49% lymphocytes. The sedimentation rate 5 mm. The Hanger test negative.

Comments: This is one case in which the correct diagnosis could have been easily missed had not Brucellosis been kept in mind. The case could have been easily confused with malaria, tuberculosis, schistosomiasis, an intra-abdominal lymphoblastoma, Hodgkin's disease or subacute bacterial endocarditis. The abnormal liver function and the liver biopsy pointed towards an acute hepatic damage. It is well known that Brucellosis involves the liver and may be at least a contributing factor in the development of portal cirrhosis. The severe low back pain suggested a Brucella spondylitis, however X Ray studies failed to reveal evidence or bone destruction. The therapy given appears to have been successful in eradicating the infection as there has been no recurrences one and a half years after discharge.

Case No. 2: A 38 year-old chauffeur from Carolina, Puerto Rico, who was admitted to San Patricio on March, 1954 complaining of fever of 10 days duration. The patient had suffered from daily fever which usually started in mid-afternoon; lasted 4-6 hours and was followed by drenching night sweats. In the mornings he had been afebrile and asymptomatic. He had developed a mild non productive cough since onset of his illness. He had lost 10 lbs. He admitted drinking raw milk from cows owned by his father.

Positive physical findings on admission were: (1) Hyperemia and swelling of nasal mucosa, (2) Hepatomegaly — the lower border of this organ being felt three finger-breadths below right costal margin, and, (3) splenomegaly — the tip of the spleen being felt one finger-breadth below left costal margin.

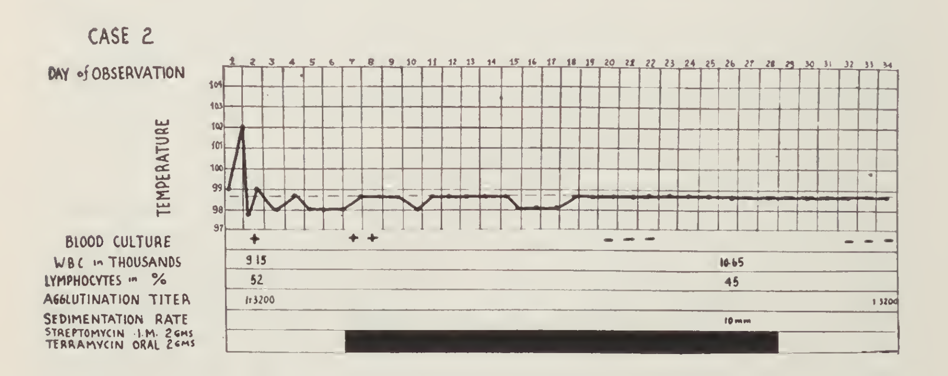
The laboratory work revealed a white cell count of 9,150. The

differential showed 2 Juveniles, 15 stabs, 31 polymorphonuclears and 52 lymphocytes. The hemoglobin was 13.5 gm. The urinalysis was normal. The Brucella agglutination titer was 1:3200. Blood cultures taken on the 2nd, 7th and 8th hospital days were positive for Brucella abortus. Liver function studies revealed a BSP retention of 10% in 45 minutes. Hanger test 3+ in 48 hours. Total Serum Proteins 7.5 gm. A/G Ratio 4.3/3.2. Studies for Schistosomiasis were negative.

Therapy with Streptomycin 2 gm. I.M. daily and Terramycin 500 mg. q.i.d. was started on the 7th hospital day and continued for 3 weeks. It should be noted that positive cultures were obtained on the 7th and 8th hospital day when the patient was afebrile and asymptomatic. Three blood cultures, 2 weeks after therapy was started, were sterile, as well as 3 cultures taken one week after therapy was concluded.

The patient was seen 6 months after discharge. He stated that one of his father's cows was found sick with Brucellosis and sacrificed 2 weeks after he was discharged from the hospital. There had been no recurrence of fever or other symptoms which would suggest active Brucellosis.

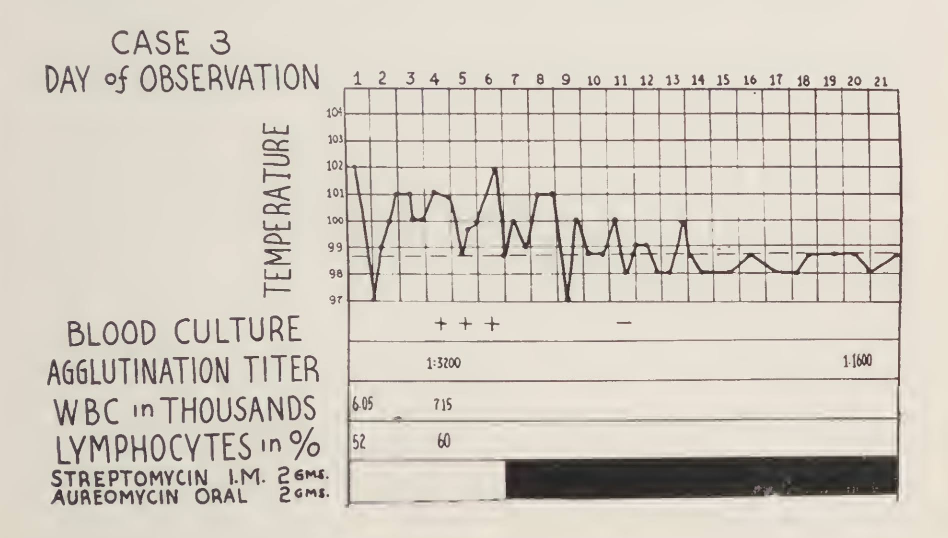
Physical examination was entirely negative. The blood culture was negative. The sedimentation rate 6.0 mm. in an hour. The Brucella agglutination titer 1:800. The white cell count 9,400 with 40% lymphocytes. The Hanger test was 1+ in 48 hours.



Comments: Here is one case who while hospitalized had fever for one day and then was afebrile and asymptomatic for the rest of the period of hospitalization. Whithout a strong index of suspicion the diagnosis could have been easily missed. The positive blood cultures in the absence of symptoms brings out the known fact that the tissues of man can be parasitized by Brucella with few or no localizing manifestations and a minimum of symptoms of ill health.

Case No. 3: A 39 year-old court secretary from Toa Alta who was admitted to San Patricio Hospital complaining of chills and fever, low back pain, headache and anorexia of 4 days duration. The chills started in the late afternoon; were followed by high fever and profuse sweats. He had received two injections of Penicillin prior to admission without any effect in his illness. He admitted drinking raw milk for years.

Physical examination was entirely negative. The laboratory work revealed a white cell count of 6,050 with 39% Polymorphonuclears, 52% Lymphocytes and 9% Eosinophiles. Examination for malarial parasites was negative. The Urinalysis was normal. The Brucella agglutination titer was 1:3200. Blood cultures taken in the 4th, 5th and 6th hospital days were positive for Brucella abortus. Therapy with streptomycin 2 gm. I.M. daily and Aureomycin 500 mg. four times a day was started on the 7th hospital day. He rapidly became asymptomatic and after two weeks of therapy was discharged with instructions to continue the antibiotics for one more week. We have no follow up in this case.



Comments: In this case there was a febrile illness of short duration with no positive physical findings. The history of drinking raw milk and the normal white cell count with relative lymphocytosis made us suspect Brucellosis. The positive blood cultures established the correct diagnosis.

It should be pointed out that the prozone phenomenon was demonstrated while performing the agglutination tests in all three cases.

In summary, we have presented three cases of Brucellosis due to Brucella abortus in which the diagnosis was confirmed by po-

sitive blood cultures. We, like others[2] have done before, urge the medical profession of Puerto Rico to be on the look-out for this disease, as we have the impression that it is more common than the scarcity of reports would make us believe. It is one diagnosis which should be considered in all cases of fever of unknown origin. Once suspected daily blood cultures should be obtained in an endeavor to isolate the offending organism. Brucellosis is one disease which can produce prolonged and debilitating symptomatology and which is amenable to therapy with the newer antibiotics.

Acknowledgement: The author is indebted to Mrs. Josefina B. Pérez, bacteriologist of this institution, for her valuable assistance in carrying out the bacteriological and serological studies.

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ACUTE HEPATIC INSUFFICIENCY IN ALCOHOLIC CIRRHOSIS

*JULIO V. RIVERA, M.D.**

In the course of diffuse liver disease profound disturbances in the various functions of the organ may occur leading to the syndrome of liver cell failure. Although a detailed physiological understanding of the metabolic disturbances operating is not yet available, the syndrome characterized by jaundice, fluid retention, bleeding tendency, profound malnutrition and central nervous system manifestations is well established. It may occur in infectious hepatitis, alcoholic cirrhosis, biliary tract disease or be due to acute hepatic necrosis. Liver failure may be precipitated by hemorrhagic shock as from bleeding esophageal varices, by severe dietary deficiency, surgical procedures performed under general anesthesia, infections and exposure to toxic agents, especially alcohol.

We are presenting three selected cases from San Patricio VA Hospital to illustrate some of the problems which arise in the management of acute hepatic insufficiency.

Case No. 1: (E.M.) The first case is that of a 37 year-old unemployed male who dated his illness to December, 1953, when while hospitalized at Juliá Clinic for an acute psychotic episode he developed anorexia and upper abdominal tenderness. The abdomen enlarged, jaundice appeared and he noted his urine to be reddish. Stools were soft and black. His family believed he was going to die and took him home on December 16, 1953. He was admitted to San Patricio VA Hospital on January 5, 1954. The history revealed a large daily intake of alcohol since 1940.

At the time of arrival the patient appeared dehydrated, markedly jaundiced and emaciated. He was tremulous and his memory was impaired. Rales were heard in the right upper lung field. The abdomen was distended with fluid. The liver was enlarged down to the umbilicus. The spleen could not be felt. The testes were atrophic. Spider angiomata were noted on the upper chest and slight palmar erythema was present. A rosette of external hemorrhoids protruded at the anus.

Hematologic study (see Figure No. 1) revealed slight anemia, marked neutrophilic (88%) leukocytosis (40,250) and thrombocytopenia (platelets 80,000 per cu. mm.). Urinalysis showed the presence of bile and increased urobilinogen (9.2 Ehrlich Units in

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two hour afternoon specimen). Prothrombin time was prolonged (23 sec., control 11 sec.). Serum albumin was 3.0 gm., globulin 3.1 gm., per 100 ml. Total serum cholesterol was 175 mg. per 100 ml. and cholesterol esters were 47%. Cephalin cholesterol flocculation was 3+ in 48 hours. The total serum bilirubin was 8.5 mgm. per 100 ml.; 6.1 mgm. indirect, 2.3 mgm. direct. Several stool specimens gave a positive guaiac reaction. Although the sputum was purulent, the culture grew no pathogens.

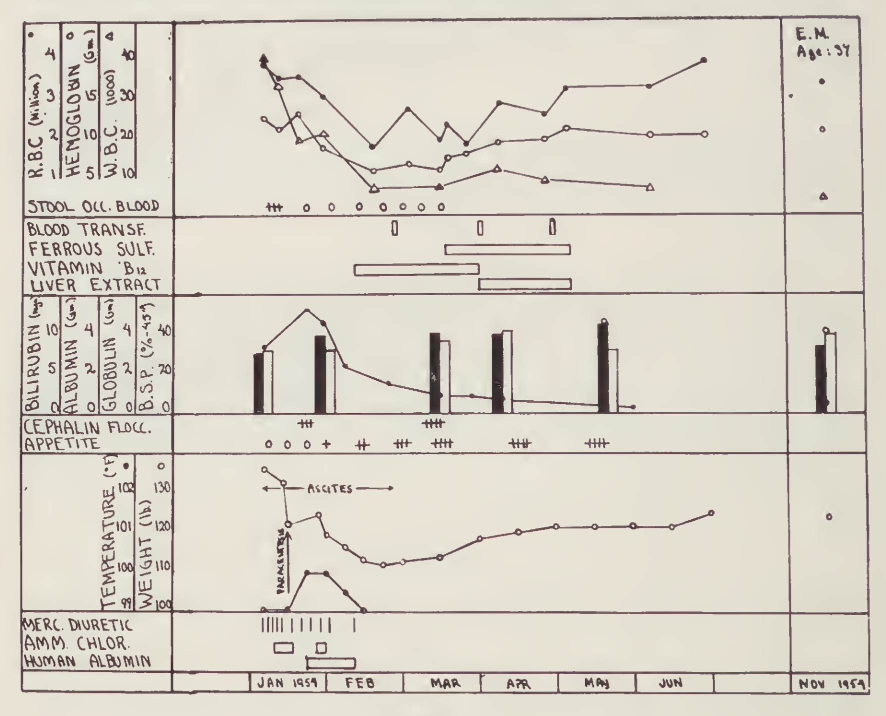


Fig. 1

Chest roentgenogram showed fibrotic changes in the right upper lobe and a small right pleural effusion. Diagnoses of alcoholic liver cirrhosis and bronchiectasis were made. Treatment was directed toward the correction of tissue dehydration and nutritional deficiencies and the control of complicating broncho-pulmonary infection. Intravenous fluids, parenteral vitamin B complex, vitamin K and penicillin were administered. A high carbohydrate, salt poor diet divided into small feedings was prescribed. In spite of our efforts the food intake at first was nil. Repeated injections of mercurial diuretics even when potentiated by ammonium chloride produced only slight diuresis. In view of our lack of success in reducing ascites and in response to the patient's complaints of discomfort, abdominal paracentesis was performed on January 15. No apparent immediate benefit was obtained from

the procedure. The food intake continued to be small, jaundice increased, fever appeared and ascitic fluid promptly reaccumulated; however, it should be noted that the body weight did not return to its previous level. On January 20, intravenous infusions of salt-free human albumin, 25 gm. daily, were started. Although the frequency of administration of mercurial diuretics had decreased, definite diuresis was evident about one week after the initiation of treatment with human albumin. It finally resulted in the total disappearance of ascites about the last week in February. At this time splenomegaly became evident. Jaundice decreased and the appetite gradually increased and later became voracious. Severe anemia became apparent as improvement of other disturbances occurred. Hematocrit studies showed the anemia to be normocytic normochromic. Bone marrow morphology was considered to be suggestive of folic acid-B_{12} deficiency. It was also noted that megakaryocytes were increased in number and platelet production seemed poor, suggesting hypersplenism. No apparent benefit followed the administration of vitamin B_{12} in adequate dosage. No evidence of hemolysis (Coomb's test, fecal urobilinogen, reticulocyte counts) could be detected. The anemia improved during the administration of ferrous sulphate, crude liver extract and blood transfusions. At the time of his last examination this patient continued to do well but he still appeared undernourished. Serum bilirubin was still slightly elevated and a mild anemia persisted. He remains unemployed.

Comment: Hepatic failure was precipitated in this patient by an acute nutritional deficiency associated with a mental disorder. Gastrointestinal bleeding and pulmonary infection may have been additional factors. Physical and laboratory examinations revealed evidence of profound alterations of liver function. Initial resistance to the action of diuretics as shown by this patient is frequently seen under these circumstances. There is in these patients delayed water diuresis and exaggerated tubular reabsorption of sodium.[2,3] This is thought to be due to the antagonizing effect of antidiuretic substances of the posterior pituitary and adrenal origin which fail to be inactivated by the diseased liver.[6] Decreased serum albumin and portal hypertension are contributing factors.[6] Although it is difficult to be certain, it appears that administration of human albumin enhanced diuresis and the subsequent improvement of this patient.

Anemia in cases of cirrhosis of the liver may be due to various mechanisms. Gastrointestinal bleeding, dietary deficiencies and hypersplenism are the most frequently mentioned. Decreased survival time of erythrocytes has also been found in some cases.[4] In this patient slow bleeding from the gastrointestinal tract was

probably the main cause. The presence of splenomegaly, thrombocytopenia and the bone marrow histology suggested hypersplenism. Multiple dietary deficiencies were probably contributory.

It is apparent that although considerable improvement has been attained by this patient, he continues to suffer from severe, chronically active liver disease.

Case No. 2: (P. M.) This 58 years-old unemployed male was admitted to San Patricio VA Hospital on January 2, 1953. In 1941 he had been treated at Mimiya Hospital for jaundice. A few years later he was hospitalized at Rodríguez Army Hospital with the same complaint. He had recovered uneventfully each time. Four months before his arrival at San Patricio VA Hospital, anorexia, jaundice and abdominal pain appeared. In the last three weeks swelling of the lower extremities and abdomen had been noted. Stools were described as light yellow. The urine was said to be red. A history of heavy alcohol intake was obtained.

At the time of admission the patient appeared drowsy, emaciated and deeply icteric. The tongue was red and atrophic. Marked ascites was present. A hard tender liver was ballotted about the level of the umbilicus. No spider angiomata, testicular atrophy or palmar erythema were found.

Anemia was not present (see Figure No. 2). Serum bilirubin was 9.6 mg. per 100 ml. Serum albumin was 2.1 gm., globulin 3.8 gm. per 100 ml. Prothrombin time was 23 seconds. Cephalin cholesterol flocculation was 4 +; thymol turbidity, 9.5 units. The urine contained bile. Serum cholesterol was 100 mg. per 100 ml; cholesterol esters 22%. The alkaline phosphatase was 4.7 modified Bodansky units.

Treatment consisted of a low salt diet supplemented by intravenous infusions of glucose and vitamins. Urinary output was at first low, but gradually increased without the aid of mercurial diuretics. Abdominal paracentesis was not required. About the end of the second week in the hospital the appetite started to improve and subsequently became excellent. Body weight decreased as the ascites disappeared but it again climbed as the nutritional status improved. The improvement in liver function is well illustrated by the change in serum cholesterol and its esterified fraction from extremely low values to within the normal range. At the time of readmission in February and September 1954 improvement was maintained although the bromsulfalein test and serum proteins showed evidence of hepatic dysfunction. In November, the patient was again hospitalized because of the recurrence of jaundice and ascites. He rapidly developed coma, convulsive seizures and died on the fifth hospital day. Post-mortem examination confirmed the clinical diagnosis of Laennec's cirrhosis.

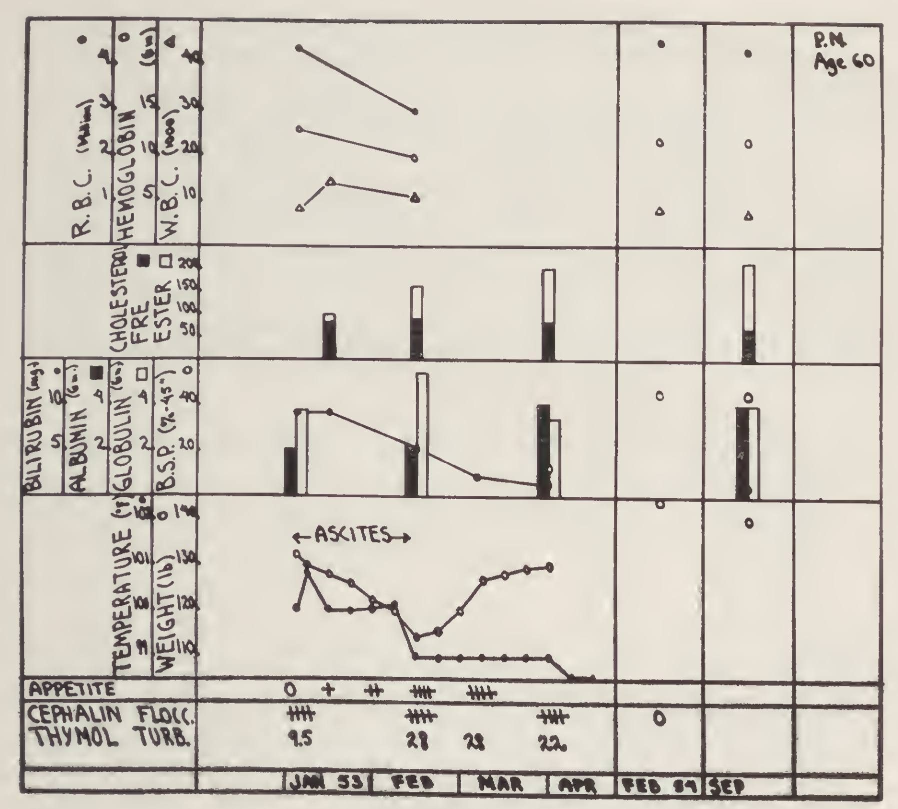


Fig. 2

Comment: The disappearance of ascites prior to the occurrence of any significant increase in the serum albumin should be noted. A low salt intake and the general improvement in liver functions appeared to be the decisive factors. This case demonstrates well the capacity of the liver to repeatedly attain considerable functional recovery even in an elderly individual. Repeated relapses resulting from the patient's inability to abstain from alcohol and maintain an adequate food intake finally led to a fatal outcome.

Case No. 3: (J.A.) This 37 year-old chauffeur was first admitted to San Patricio VA Hospital on June 23, 1952 with complaints of abdominal swelling and jaundice of recent onset. He gave a history of prolonged alcoholism. Because of the development of delirium tremens the patient had to be transferred to Juliá Clinic where he remained until July. 9. While at that institution his mental symptoms improved but his physical condition did not. He was again transferred to this hospital.

At the time of readmission the patient appeared markedly undernourished and deeply jaundiced. Numerous spider angiomata were present on the upper part of the body. Sacral and leg edema

were noted. Numerous ecchymoses were present at the sites of venipuncture. The skin was pale and atrophic. The abdomen was distended with fluid. A hard, non tender liver edge was felt five centimeters below the costal arch. The spleen was not felt.

The hemogram (see Figure No. 3) was as follows: RBC, 1,200,000 per cu. mm.; hemoglobin, 5.8 gm. per 100 ml.; hematocrit, 27.5% ; MCV, 229 cu. microns; MCH, 48.3 micro-micrograms; MCHC, 21.1% ; WBC 20,600 per cu. mm., 86% neutrophils, 6% lymphocytes, 4% monocytes; platelets 125,000 per cu. mm. Reticulocytes were 0.2%. Bone marrow showed hyperactivity of all elements and megaloblastic development of some erythrocyte precursors.

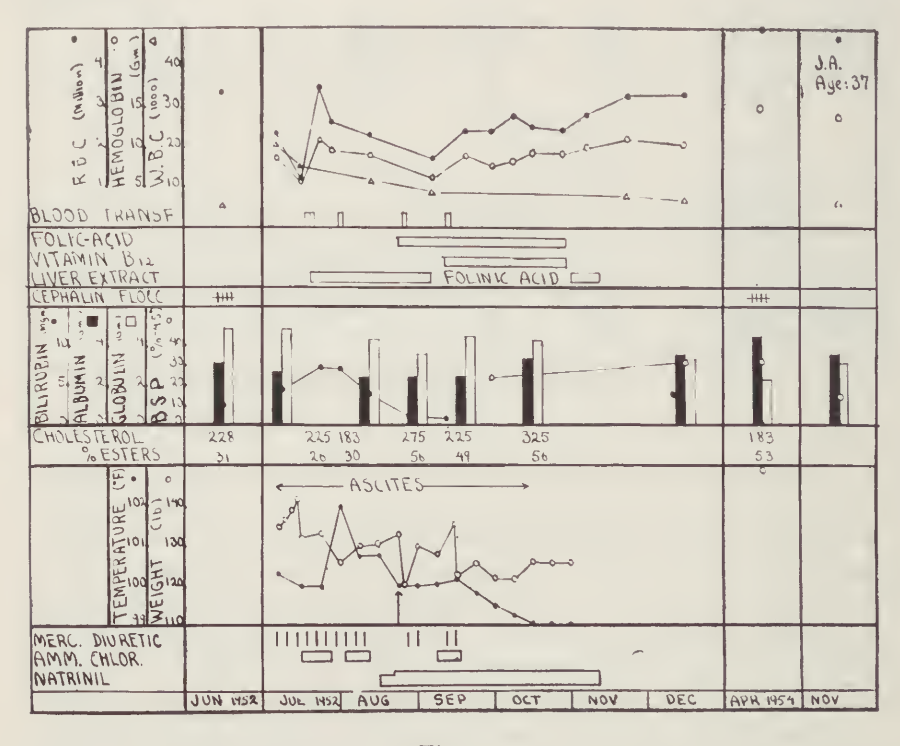


Fig. 3

The serum bilirubin was 3.9 mg. per 100 ml. Prothrombin time was 19 seconds; control, 11 seconds. Serum albumin was 2.7 gm., globulin, 4.9 gm. per 100 ml. Cephalin cholesterol flocculation was 4 +. Total serum cholesterol was 225 mgm. per 100 ml., cholesterol esters 26%.

Response to mercurial diuretics and ammonium chloride was irregular. In view of this a cation exchange resin. (Natrinil)R was added to the therapeutic regime. Since no apparent benefit was obtained, abdominal paracentesis was performed on August 23.

Fluid reaccumulated rapidly during the following week. During the last two weeks of August, fever and jaundice decreased and the prothrombin time and proportion of cholesterol esters approached normal. By mid-September a response to mercurial diuretics was again obtained resulting in a decrease in ascites; however, the anemia did not improve. Repeated epistaxes and bleeding from the paracentesis site aggravated it. Because of its profound degree blood transfusions were given. Therapeutic trials with crude liver extract, folic acid, vitamin B_{12} and folinic acid did not produce a reticulocyte response. Anemia gradually improved over the next few months as the nutritional status of the patient bettered. Following discharge from the hospital the patient returned to his usual occupation and he has remained gainfully employed during the following two years. He has abstained from the use of alcohol. At the time of his last examination he was asymptomatic; however, hepatosplenomegaly is still present and extensive varicosities of the lower esophageal veins were demonstrated on endoscopic and roentgen examination.

Comment: Macrocytic anemia has been described not infrequently in cirrhosis of the liver but it is only rarely accompanied by a megaloblastic bone marrow.[5] This anemia may be related to impaired ability of the hepatic tissue to store the erythrocyte maturating factors or perhaps to protein deficiency.

This case exemplifies to what extent rehabilitation is possible in this disease when the patient is able to make the necessary psychological readjustments. Cirrhosis is now clinically latent in this patient. He may well remain in this asymptomatic state indefinitely. On the other hand, the threat of the effects of portal hypertension with rupture of esophageal varices remains.

SUMMARY

Some of our experiences in the management of acute hepatic insufficiency in cases of alcoholic cirrhosis have been related. These cases have exhibited various problems which are encountered in this condition. A flexible therapeutic regime directed towards the correction of the various metabolic derangements presenting has been employed. The importance of correcting the multiple nutritional deficiencies and water and electrolyte disturbances is emphasized. The tremendous capacity of the liver for functional recovery is demonstrated.

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THE PROBLEM OF THE COIN LESION

*ALFRED L. AXTMAYER, M.D.**
*LASZLO EHRLICH, M.D.***

Surgical Aspect:

Much has been said in recent years about the so-called coin lesion of the lung. One of these days you will be confronted by the problem of a friend, relative, or even yourself, who just had a routine chest X-ray and that X-ray film reveals a coin lesion. What will you advise your patient or what will you do yourself?

By coin lesion we refer to the single circumscribed lesion which causes no distal obstructive changes in the lung.

There are two beliefs which have been responsible for the deeply ingrained conservatism shown by the majority of physicians when this type of lesion is encountered. These are the concepts that these lesions can be safely watched; and that thoracotomy is a formidable procedure that carries with it a high degree of morbidity and a high mortality rate.

The purpose of this presentation of the problem of the coin lesion is to attempt to destroy these concepts and to show you why we feel that they are wrong.

The concept of high morbidity and mortality of thoracotomy has been completely disproven in recent years. Large series of cases reported by the great medical centers of the U. S. have shown that thoracotomy can be performed with mortality rates well under 1%.

In our own hospital we have performed over 100 purely thoracic surgical procedures since 1949 with only 2 operative deaths.

The establishment of a definite unmistakable diagnosis of all abnormal lesion in the thorax is imperative, regardless of whether or not the patient has any symptoms referable to it.

The solitary lesion of the lung is one that eludes absolute certain diagnosis no matter what diagnostic procedure is employed. The use of X-rays including specialized techniques such as tomography and bronchography, as it will be demonstrated has many short comings. Many different lesions in various locations have very similar roentgenologic manifestations and a definite diagnosis cannot be established by these means. Dr. Ehrlich will show that the X-ray characteristics of a given lesion can be very misleading.

Bronchoscopy has been of little help except in those rare oc-

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Presented at the Annual Meeting of the Puerto Rico Medical Association, December 10, 1954.

casions where the lesion is centrally located and involves a major bronchus.

The patient's symptomatology can be very misleading, hemoptysis being about the only symptom that we view with concern in these cases.

Routine laboratory studies are of little use except that elevation of the erythrocyte sedimentation rate is common in malignant disease.

The tuberculin test is only of value when it is negative, helping us rule out tuberculosis as a cause of the lesion.

Once these available diagnostic methods are exhausted and no definite diagnosis arrived at it is of the utmost importance that early thoracotomy be performed. Exploratory thoracotomy is justifiable because:

a) The risk is slight.
b) The lesion may be malignant.
c) If the lesion is benign it is potentially dangerous to life.

At thoracotomy the exact diagnosis can frequently be made with ease by the gross appearance of the lesion and the surrounding lung, lymph nodes and other structures. If in doubt frozen section study is available and should be used.

It is important that the lesion be completely removed in a one stage operation. If benign the operation should be as conservative as possible, the extent of the procedure to be determined by its location. If malignant, surgery should be as extensive as necessary for adequate removal of the primary lesion and any metastases.

We therefore present the only rational plan for the treatment of the solitary mass in the lung.

First—X-ray picks up the lesion and the roentgenologist is the first to be in a position to offer advice.

Second — No matter why the X-ray was taken, be it as a routine mass survey or because of some symptoms, exploratory thoracotomy should be immediately recommended except where the lesion is calcified and therefore practically always benign and where other physical factors make operative risk prohibitive.

Therefore, let us not be at fault by being too conservative. The tragedy of the inoperable tumor, be it an advanced benign tumor that has degenerated and becomes malignant or that has grown large enough to infringe and interfere with neighboring structures, or be it a malignant tumor that has become incurable, is too well known to warrant debate.

We, therefore, must insist on the recommendation of thoraco-

tomy for the adult patient with the solitary lung lesion. We make only two exceptions to this statement:

a) Absolute medical contraindication to major surgery.

b) The highly calcified small solitary lesion which is practically always a tuberculoma.

Radiological Aspect

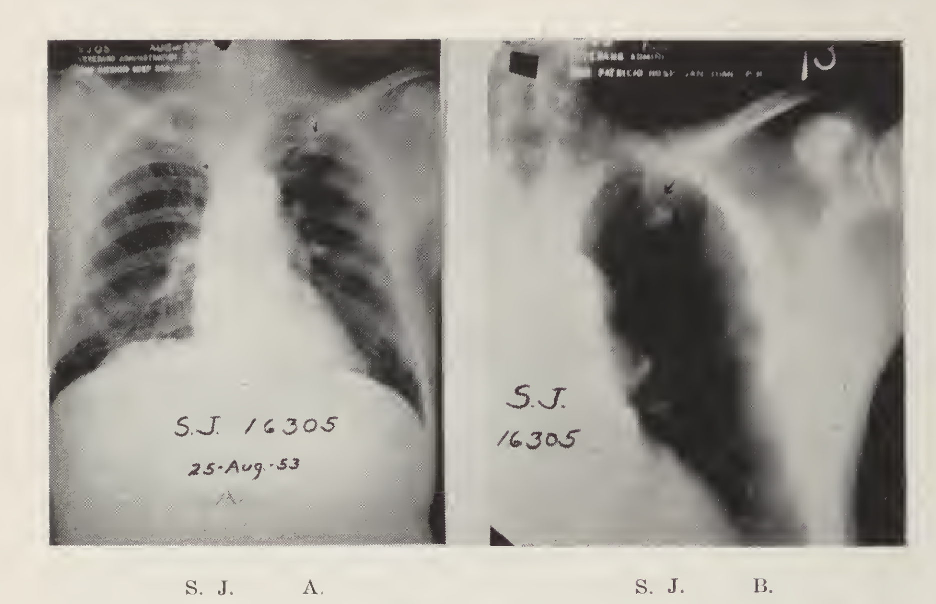
The coin lesion, as an X-ray term, refers to a roundish density, ranging from a nickel to a silver dollar, usually located in the periphery of the lung fields, surrounded by clear parenchyma and usually picked up on a routine chest film. The immediate problem is to determine whether this finding is a benign lesion or a carcinoma.

Among the benign conditions, we usually consider Tuberculoma or some rare possibilities, such as Hamartoma, Hemangioma and Adenoma.

Now, a few years ago, I would have greatly enjoyed presenting a detailed discussion concerning the differential diagnosis. Lately, we have learned, that all the previously believed criteria, such as size, shape, sharpness of borders, evidence of growth, are of no value. At the present time, the only criterium of benignity that can be relied upon with reasonable certainty is the demonstration of calcification in the lesion.

In other words, the duty of the roentgenologist begins and ends by reporting the coin lesion, emphasizing the presence or the absence of calcification in it. It is the prerogative of the clinician to make the final decision regarding operation.

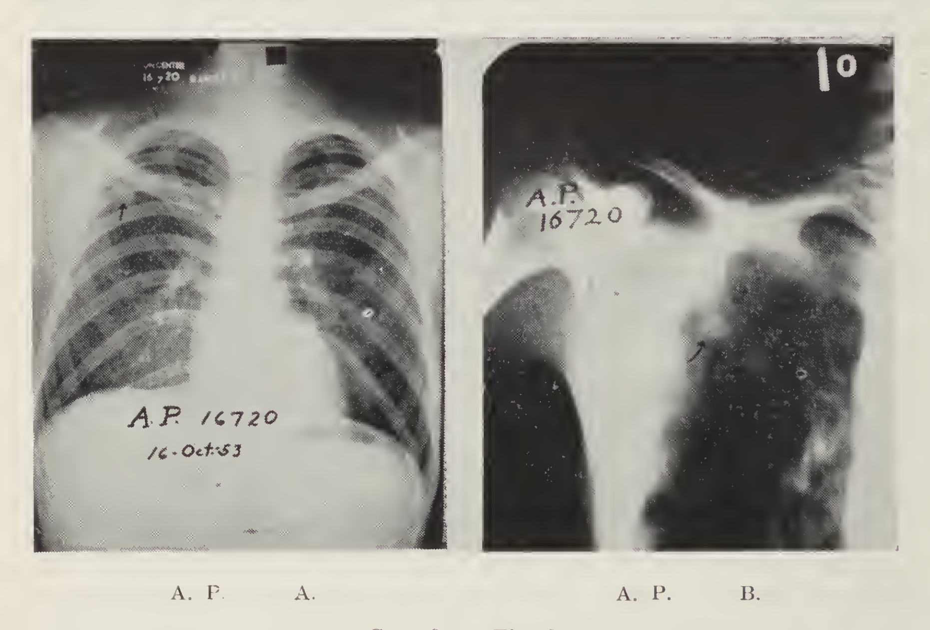
Now, let me show you our small series of 9 proven cases.



S. J. A. S. J. B.

Case 1. Fig. 1.

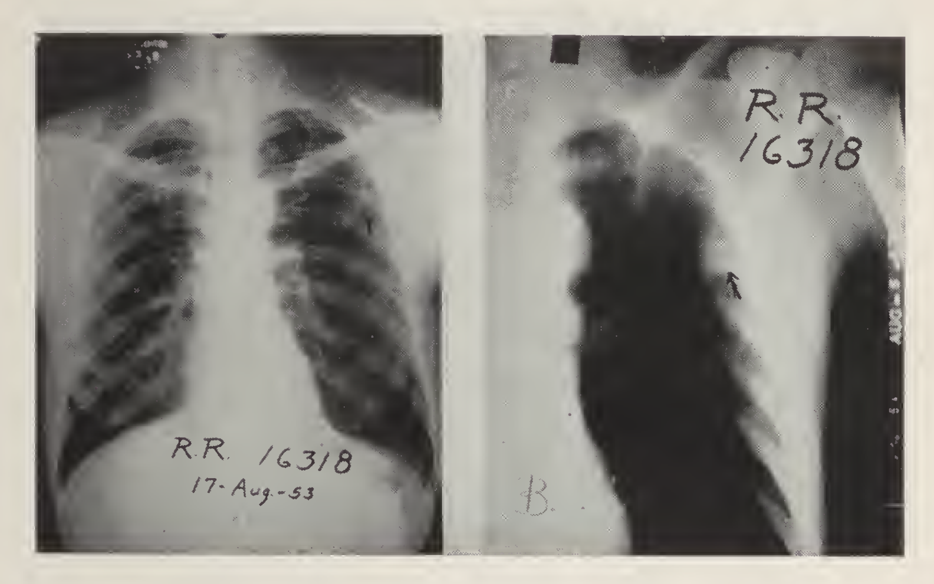
S. J.—Age: 70. Hypertensive. Routine chest (A): Ovoid lesion of 2 cm. under left first rib. Laminography (B): shows calcification. Impression: Tuberculoma. Op.: Wedge resection. Path: Tuberculoma. Pt. is well 11 months post-op.



A. P. A. A. P. B.

Case 2. Fig. 2.

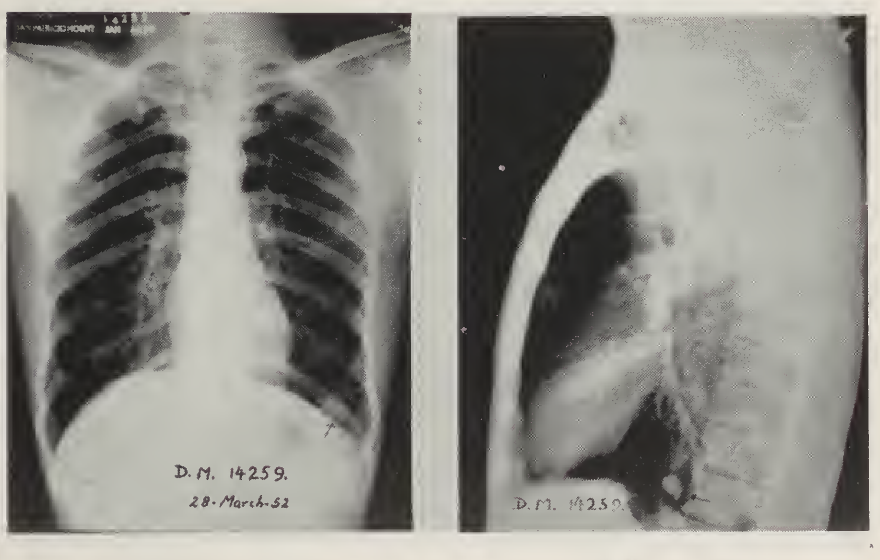
A. P.—age: 33. Pterygium. Routine chest (A): Ovoid lesion of 1.2 cm. in rt. 2nd ant. space. Laminography (B): No calcification. Impr.: Tuberculoma. Op.: Wedge resection. Path: Tuberculoma. Pt. is well 14 months post.-op.



R. R. A. R. R. B.

Case 3. Fig. 3.

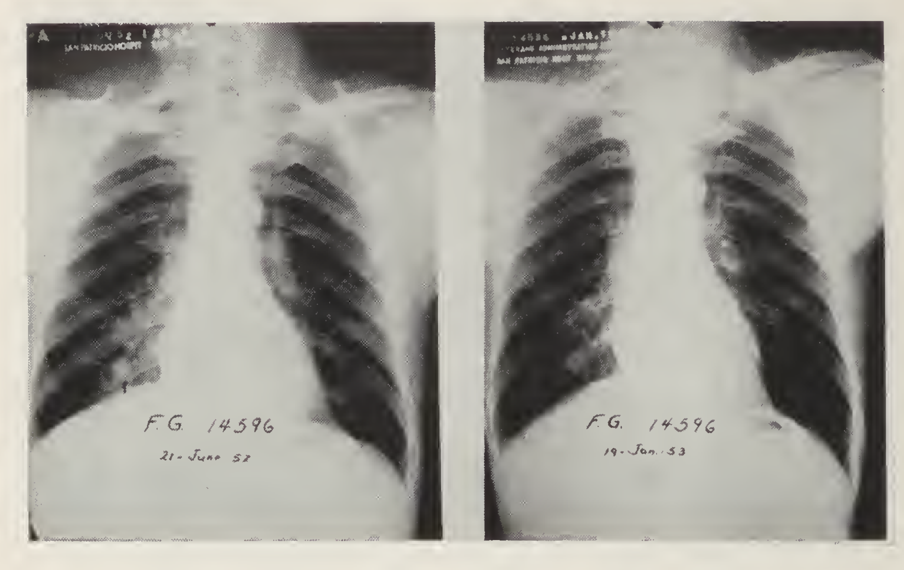
R. R.—Age: 61. Left chest pain for 4 months; non-productive cough; wt. loss of 19 lbs. Chest film (A): Oblong lesion of 25 x 18 mm. in LUL. Laminography (B): no calcification. Impr.: Tuberculoma vs. CA (Ca favored.) Op.: Segmental resection. Path:: Tuberculoma. Pt. is well 15 months post-op.



D. M. A. D. M. B.

Case 4. Fig. 4.

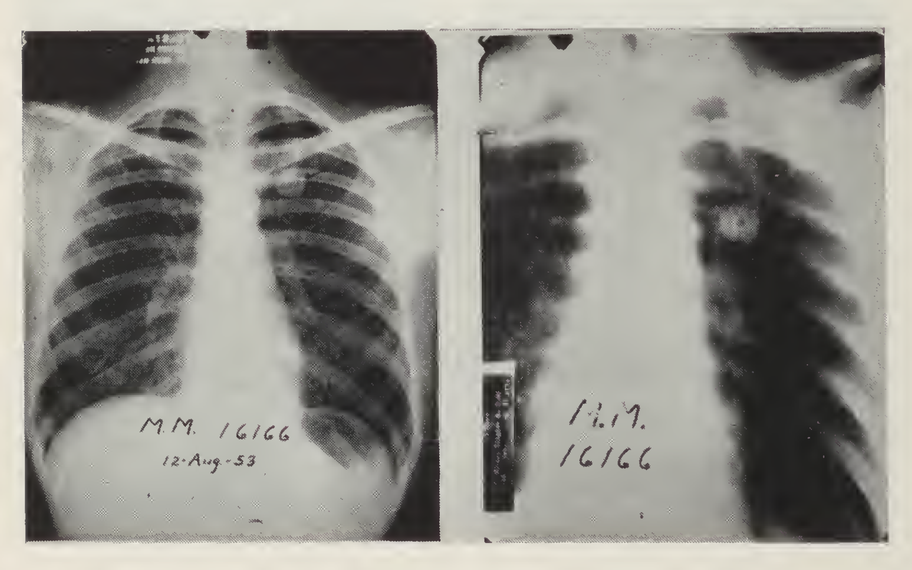
D. M.—Age: 30. Peculiar sensation in the left chest for 6 years; question of bruit in left lower chest heard by some. Chest film: Ovoid lesion of 25 x 15 mm. in LLL. Impr.: A-V aneurysm vs. Tuberculoma (Tuberculoma favored). Op.: Wedge resection. Path.: Tuberculoma. Pt. is well 23 months post-op.



F. G. A. F. G. B.

Case 5. Fig. 5.

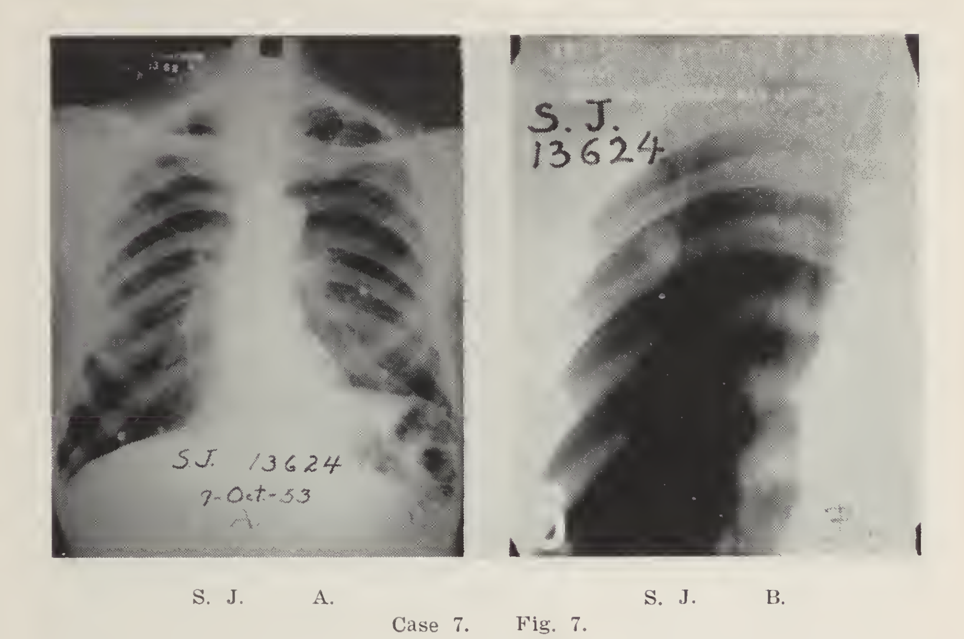
F. G.—Age: 55. Prostatic hypertrophy. Routine chest (A): Neg. (Note obvious lesion at rt. base). Readmission 6 months later for cough and bloody sputum; loss of wt. of 14 lbs. Chest film (B): Thick-walled cavity at right base. Impr.: Diag. is uncertain. (Bronchiectasis vs. Tb.) op.: Lobectomy. Path.: Tuberculoma. Pt. is well 21 months post-op.



M. M. A. M. M. B.

Case 6. Fig. 6.

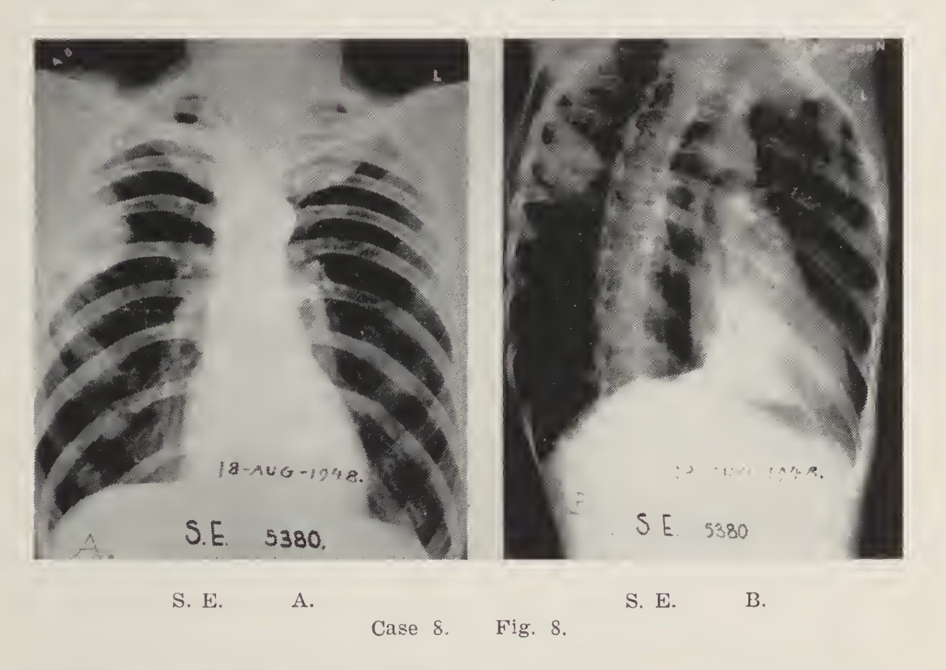
M. M.—Age: 29. Migratory chest pain for 1 year. Chest film (A): Round lesion of 2.5 cm. in LUL with cavitation in its center. Laminogram (B): Calcification present. Impr.: Tuberculoma and other smaller tuberculomas surrounding it. Op.: Segmental resection. Path.: Tuberculoma with satellite nodules. Pt. is well 17 months post-op.



S. J. A. S. J. B.

Case 7. Fig. 7.

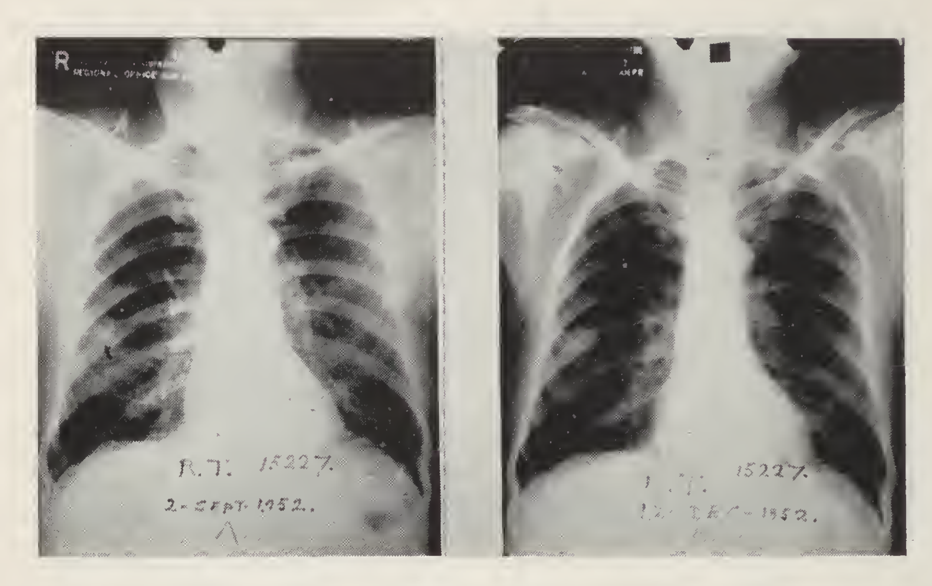
S.J.—Age: 63. Operated for a Ca of tongue 2 years before. Routine chest (A): Round lesion of 1.5 cm. in RUL. Laminogram (B): No calcification; central necrosis. Impr.: Tuberculoma, with central "caseation". Op.: Wedge resection. Path.: Metastatic Ca. Pt. is well 14 months post-op.



S. E. A. S. E. B.

Case 8. Fig. 8.

S. E.—Age: 64: Productive cough with mild fever for 9 months. Chest film: Roundish lesion of 4 cm. in RUL. Impr.: Tumor. Op.: Pneumonectomy. Path.: Bronchogenic Ca. Pt. is well 6 years and 4 months post-op.



R. T. A. R. T. B.

Case 9. Fig. 9.

R. T.—Age: 66. Seen at the Regional Office for pension. Routine chest (A): Ovoid lesion of 2 cm. in rt. chest. Impr.: Tuberculoma. Admitted to San Patricio 3 months later for operation. Chest film (B): The lesion has increased in size. Impr.: Probable Ca. Op.: Lobectomy. Path: Bronchogenic Ca. Pt. is well 24 months post-op.

PARTIAL OBSTRUCTION OF THE PYLORUS DUE TO A MUCOSAL DIAPHRAGM OF CONGENITAL ORIGIN

*LUIS A. PASSALACQUA, M.D., F.A.C.S.***
*CALIXTO A. ROMERO, M.D.**

Three years ago, a report of an extremely rare gastric lesion appeared in the British literature: (1) The patient was a 40 year old woman who had an ulcer of the lesser curvature of the stomach, symptomatic for at least 6 years. She came to operation, after 2 years of vomiting whenever she ate large meals, and was found to have, in addition to the ulcer, a mucosal diaphragm separating the body of the stomach from the antrum. This mucosal diaphragm had a small eccentric opening which communicated the body and the antrum of the stomach. The clearly defined and regular structure of this diaphragm unassociated with excess gastric rugae suggested to the author a congenital rather than an acquired condition. We have encountered a similar mucosal diaphragm giving rise to persistent symptoms of partial pyloric obstruction in a patient past middle life. The purpose of this report is to record a second variant of the unusual deformity reported by Sames, and to aduce further evidence of the probable congenital basis for such anomaly.

CASE REPORT

A 62 year old painter was admitted to the Veterans Administration Hospital on 2-27-50, complaining of epigastric pain and occasional vomiting after meals. Following the extraction of some 16 teeth, as treatment for pyorrhea alveolaris, twelve years previously, he commenced to experience dull non-radiating epigastric pain one half hour after meals. This pain, which lasted from 10 to 30 minutes, was sometimes accompanied by a feeling of fullness in the upper abdomen and frequently by nausea. At times it let to regurgitation of the ingested food and consequent relief of all symptoms. In 1947 the first upper G.I. roentgenograms performed were reported normal. A second G.I. series performed at a VA Contract Hospital revealed slight barium residue, 6 hours after the barium meal, in an otherwise normal stomach. Shortly after-

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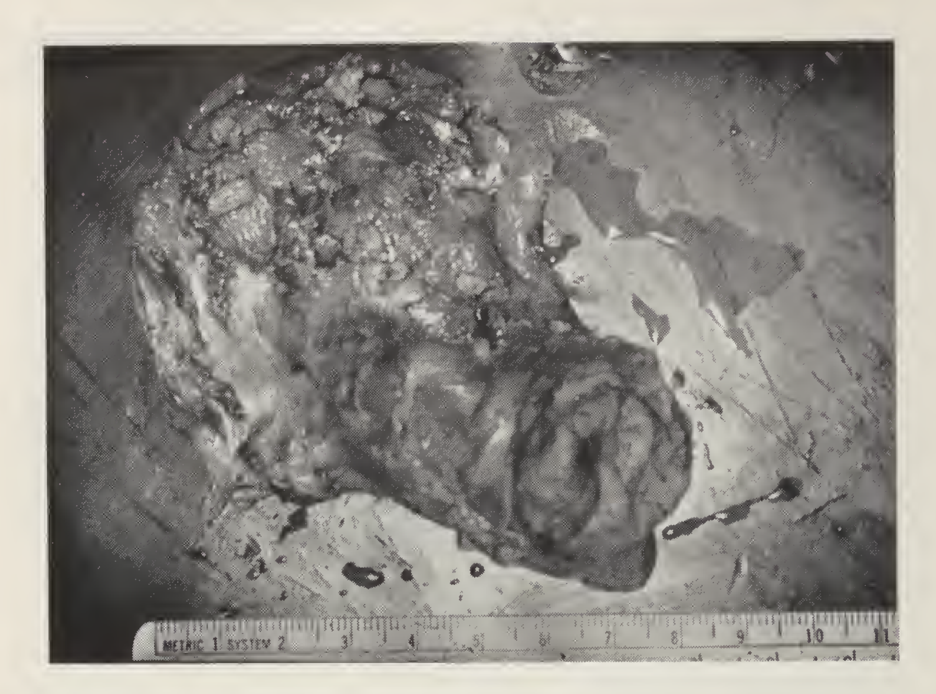


Figure No. 1 — ATRESIA OF PYLORUS WITH PARTIAL OBSTRUCTION
GROSS: Stomach, subtotal, measuring 5 cm. along the lesser curvature by 12 cm. along the greater. The lumen of the organ has been exposed and it shows a slight dilatation of the pyloric antrum or pars pylorica of the stomach associated with a severe atrophic gastritis. The atrophic changes are milder in the pars corpus of the organ. The pylorus (pyloric sphincter) presents a defective development of the musculature which has resulted in relaxed or dilated pyloric aperture about 2 cm. in diameter, almost closed by a diaphragmatic mucosal membrane with an eccentric roundish opening 6 mm. in diameter. The opening is displaced upwards and anteriorly. This anomaly has lead to formation of a recess or pouch downwards and posteriorly between the pyloric antrum and duodenum. The duodenal segment measures between 0.5 and 2 cm. in length. The duodenal mucosa shows marked atrophic changes and erosion.

wards in October 1949, he underwent an exploratory laparotomy. An apparently normal stomach was opened longitudinally along its lower third and a seemingly normal gastric mucosa was exposed. The pylorus appeared contracted but a rubber size 16 French catheter was passed snuggly into the duodenum. No other pathology was discovered on inspection of this area. There was no change in patient's symptoms following operation. Post-operative G.I. x-rays failed to disclose any significant abnormalities. Because of the persistence of symptoms the patient returned in 1950.

Physical examination was not contributory. Urinalysis, blood serology, CBC and coprologic studies for ova and parasites were within normal limits. Free hydrochloric acid, up to 23 clinical



Figure No. 2 — ATRESIA OF PYLORUS WITH PARTIAL OBSTRUCTION

MICROSCOPIC: The *gastric mucosa* pertaining to the pars pylorica shows severe atrophic changes with almost total disappearance of its glandular structures, extensive erosion and superficial ulceration and a heavy lymphocytic infiltrate of the remaining lamina propria, which extends into the superficial portion of the submucosa in the form of lymphoid follicles. The gastric mucosa pertaining to the body shows mild atrophic changes associated with lymphocitic, neutrophilic and eosinophilic infiltrate of the lamina propria. The *duodenal mucosa* is markedly atrophic, eroded over extensive areas and superficially ulcerated over smaller ones; its lamina propria is infiltrated with lymphocytes, plasma cells and fewer eosinophiles.

units, was obtained from the stomach one half hour after histamine injection. X-ray studies disclosed the presence of a persistent deformity of the body of the stomach at the site of previous gastrostomy. On gastroscopy, the antrum was readily visualized but the pyloric canal did not come into view; the mucosa of the stomach was of normal appearance. One sample of gastric contents studied by the Papanicolau method was reported negative for malignant cells. Exploration was recommended once again. This time however, the pyloric region was carefully inspected after opening the stomach and the curious anomaly noted before was again encountered. The pyloric canal was obstructed by a mucosal diaphragm which had an eccentric rounded opening 6 mm. in diameter (Fig 1). The duodenum beyond appeared relaxed; it measured 2 cm. in diameter and exhibited a small recess or pouch along its greater curvature side. A subtotal resection of the stomach and first portion of the duodenum was performed.

Close inspection of the specimen submitted for pathologic examination showed no other abnormalities in the stomach or adjacent duodenum. The recess in the duodenal side corresponded in position to the portion of the duodenum which bore the brunt of the gastric contents propulsed by peristaltic activity through the eccentric small opening in the mucosal pyloric diaphragm. Section through the pylorus disclosed that there was no change in the thickness of the gastric musculature in the region of the pyloric ring. Microscopic section through this area (Fig. 2) revealed hypoplasia or atrophy of the muscle of the pyloric sphincter. The mucosal diaphragm consisted of a double layer of mucosa, muscularis mucosae and submucosa. The duodenal side or the membrane contained characteristic duodenal submucosal glands. The gastric side of the membrane included glands of the gastric type only. The mucosa of the antrum was thinned out and diffusely infiltrated by moderately abundant lymphocytes and scattered plasma cells.

DISCUSSION

Whether congenital or acquired, the type of anomaly exhibited by this patient would appear to be extremely rare. If acquired we must consider it as due to redundancy and prolapse of the mucosa with partial obstruction of the pylorus. We feel, however, that several considerations weigh heavily against such a possibility. In the first place, prolapse of the gastric mucosa is seldom if ever associated with persistent symptoms of obstruction. Intermittent obstructive symptoms are said to occur in rare cases of extreme prolapse into the duodenum.[2] Roentgenographically there were no changes suggestive of mucosal prolapse such as have been described.[3] The pathologic examination yielded further evidence against the possibility that the mucosal diaphragm could be due to an artefact of mucosal redundancy. The lumen of the pyloric canal narrowed by mucosal prolapse must be of stellate cross section because the mucosa corresponding to a larger viscus, the stomach, is crowded through a narrow opening and consequently plicated longitudinally. The opening of the mucosal diaphragm described above was rounded. Furthermore, in the fresh specimen, mucosal prolapse is characterized by redundancy of the antral mucosa and reductibility of the prolapse features not encountered in our specimen. The fifth bit of evidence against this etiology was obtained from the microscopic examination. A fold of gastric mucosa prolapsing through the pylorus would not be expected to include duodenal submucosal glands as were observed in this diaphragm.

We believe that the stenosis of the pylorus encountered in

this patient is of congenital origin. Even though the patient stated afterwards that he had been subjected to stomach aches and occasional vomiting during childhood and easily whenever he operate, the age of onset of symptoms is an argument against this contention. It is noteworthy that the condition became apparent clinically after multiple tooth extractions which rendered the patient unable to masticate his meals properly. It would seem reasonable to conclude that the condition could have existed before the onset of symptoms and not become clinically manifested because the food was being thoroughly masticated. It should be noted also, that obstructive symptoms existed for twelve years and that during this time they did not progress appreciably as one would expect in the case of an acquired pyloric obstruction. Our thesis for the congenital nature of the lesion is further supported by the hypoplasia of the pyloric musculature observed in association with the mucosal diaphragm, and by the other anatomic features discussed above as evidence against a mucosal prolapse.

Arey takes cognizance of the existence of atresia of the pylorus[4] but does not cite references nor does he state whether this condition has been observed in the human after birth. We have not been able to find any similar cases other than the one reported by Sames. The finding of a mucosal diaphragm in our case as well as in that reported by Sames is reminiscent of the transient epithelial occlusion that occurs in the human embryo before the seventh week throughout the intestinal tract. The persistence of an epithelial membrane at different levels of the gastrointestinal tract explains the phenomenon of atresia, partial or complete, that is generally recognized.

SUMMARY

1. A case of partial obstruction of the pylorus due to a mucosal diaphragm is described.

2. Evidence is brought forward to show that the rare lesion described in all probability represents a congenital atresia of the pylorus.

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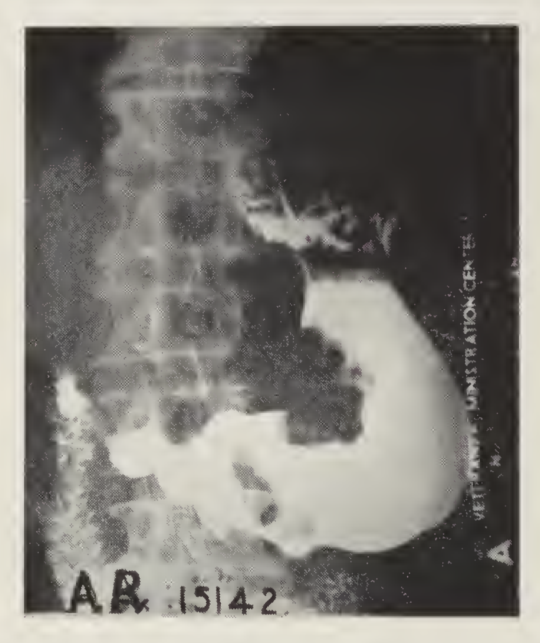
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TECHNICAL ADVANCES IN THE ROENTGENOLOGICAL DEMONSTRATION OF CARCINOMA OF THE STOMACH.

*LASZLO EHRLICH, M.D.**

The title of this presentation may be somewhat misleading, because nothing significant has been invented in the way of technique by us or by anybody else. What I would like to convey, could be best expressed by an example. Take for instance an X-ray examination of a lumbar spine; the chances are, that 8 or 9, out of 10 equally well-trained radiologists, will make the same diagnosis. Why? Because the technically acceptable A-P & lateral views of the lumbar spine are standard views, offering exactly the same amount of information. This is not so in the case of the x-ray examination of the stomach. Take again the same 10 equally well-trained radiologists and have them examined the same stomach, you will have different examinations. So different and characteristic, that you can almost recognize the radiologist from the films taken by him! The human factor is at its maximum in the fluoroscopy of the stomach.



Case 1. (Fig. 1) A.R.—Age: 75. Epigastric distress and p.c. vomiting for 2 months; wt. loss of 22 lbs. - Clinical diag.: CA of stomach. X-ray diag: CA of stomach (obvious). - Op.: inoperable CA.

* Chief, X-Ray Service, San Patricio V.A. Hospital.
Presented at the Annual Meeting of the Puerto Rico Medical Association, December 10, 1954.

Now, I believe, that a reappraisal of the general approach to the examination of the stomach is the most important advance in technique, and particularly so in the roentgen demonstration of a Ca. and of the stomach.

When you have a case like this (Fig. 1), regardless the technique used, the diagnosis of Ca. is almost written on the film.

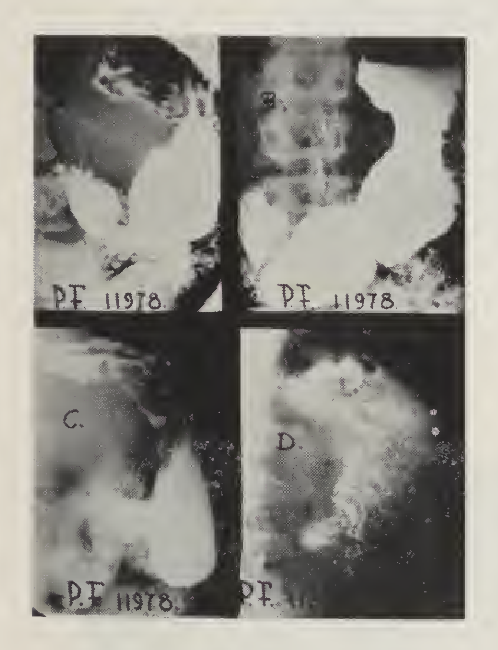


Fig. 2

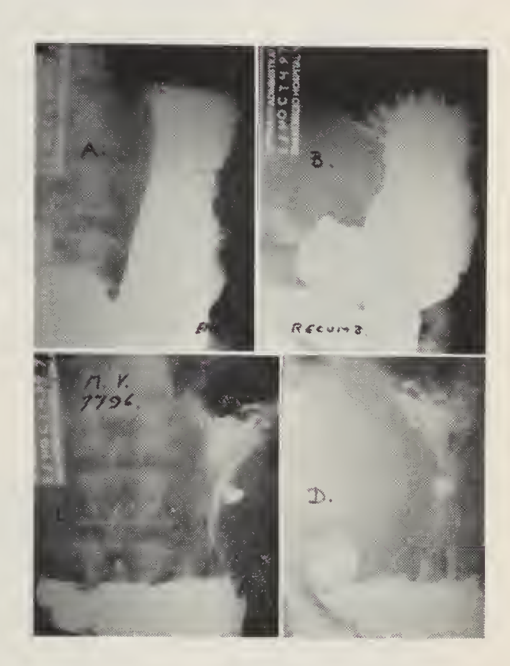


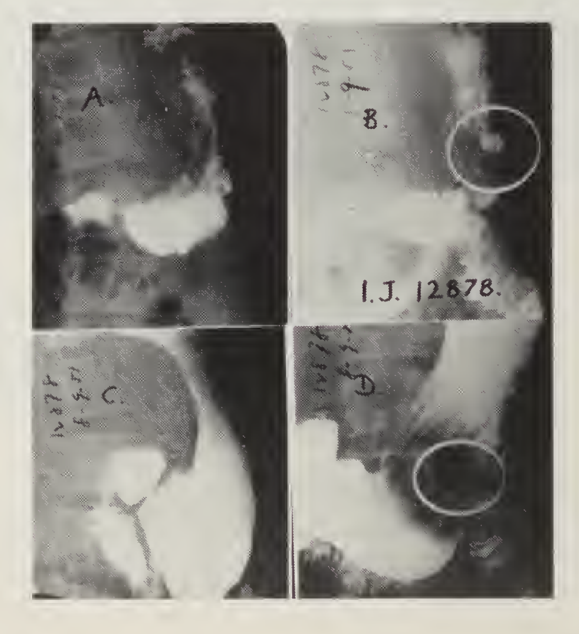
Fig. 3

Case. 2. (Fig. 2) P. F.—Age: 64. Constant epigastric pain and massive hematemesis. - Cl. diag.: esophageal varices vs. ulcer. - X-ray diag.: CA at lesser curvature. - Compare views of barium-filled stomach, recumbent (A), and erect (B) with mucosal study (C) showing the irregularity. - Op.: total gastrectomy (B). Path.: CA of stomach, with metastatic involvement of the nodes. - Pt. is alive 26 months post-op.

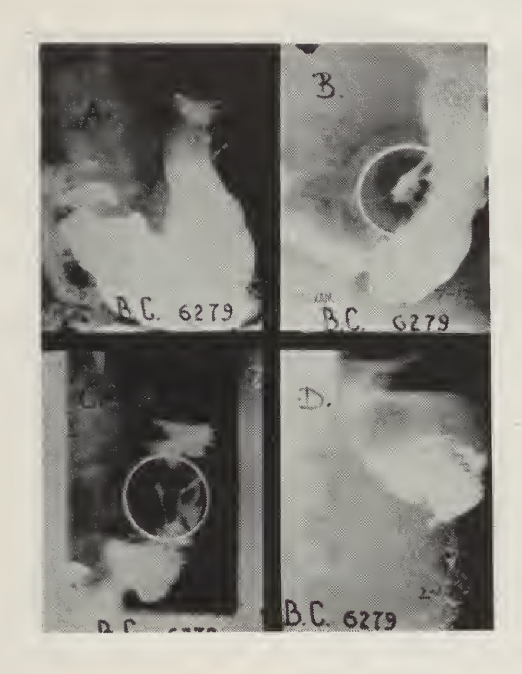
Case 3. (Fig. 3) M. V.—Age: 59. Vague epigastric pain for 6 years. - Cl. diag.: Gastritis. - X-ray diag.: Gastric ulcer, at greater curvature, malignant. - Compare views of barium-filled stomach, erect (A) and recumbent (B) with mucosal studies (C&D), showing the crater. - Op.: Subtotal. Path: Benign ulcer.



Case 4. (Fig. 4) S.C.—Age: 64. Epigastric discomfort for 6 months - Cl. diag.: "Rule out CA." - X-ray diag.: Rigidity of lesser curvature of antrum; gastroscopy advised. - Gastroscopy: Neoplasm. - Compare recumbent views (top row) showing rigidity better than the erect views. Op.: Subtotal. - Path.: CA (perhaps secondary becuse of non-involvement of the overlying mucosa.) Pt. is alive 4 years and 7 mo. post-op.



Case 5. (Fig. 5) I. J.—Age: 32. Epigastric pain for 2 years; laparotomy for perforated ulcer 1 year before this admission. Cl. diag.: D. U.-X-ray diag.: Disturbed mucosal pattern; 2 craters seen fluoroscopically that could not be consistently reproduced on films. Impr.: Adhesions, due to previous operative procedures. Compare mucosal study (A) and small crater under palparium compression (B) with barium filled stomach (C), and under compression (D) showing no ulcer.



Case 6. (Fig. 6.) B. C.—Age: 69. Constant dull epigastric pain for 6 months. Cl. diag.: CA of stomach. - X-ray diag.: Flat crater seen only under compression, probably malignant. Re-examination advised. Compare (A) with (B). Re-examination (by a different radiologist) (C): Disturbed mucosa at lesser curvature; the previously present crater is not identified; lesion is probably malignant. - Op.: Subtotal (D). Path: CA in situ. - Pt. is well 3 years post-op.

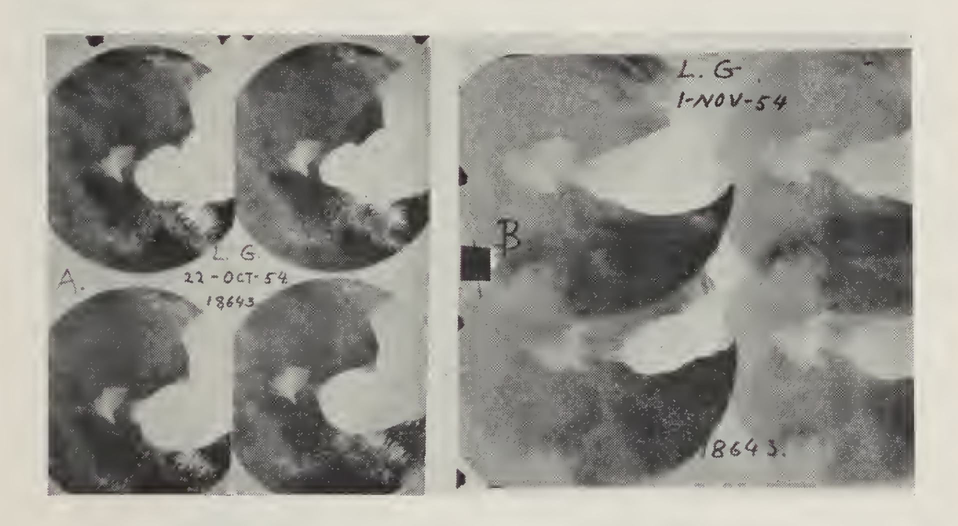


Fig. 7 Fig. 8

Case 7. (Fig. 7, 8 & 9) L. G.—Age: 66. Epigastric pain for several years. Cl. diag.: "R. O. pathology". - Three different examinations by three different radiologists. - First exam. (not shown): "Narrowed prepylorus with mucosal changes which should be investigated in order to rule out CA." - Second exam.

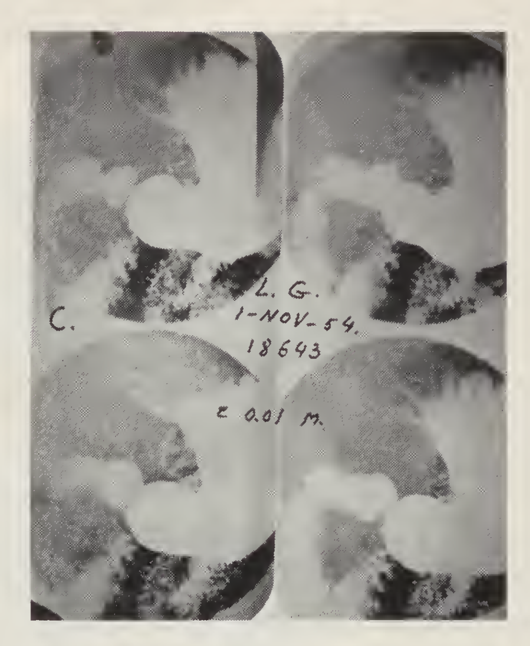
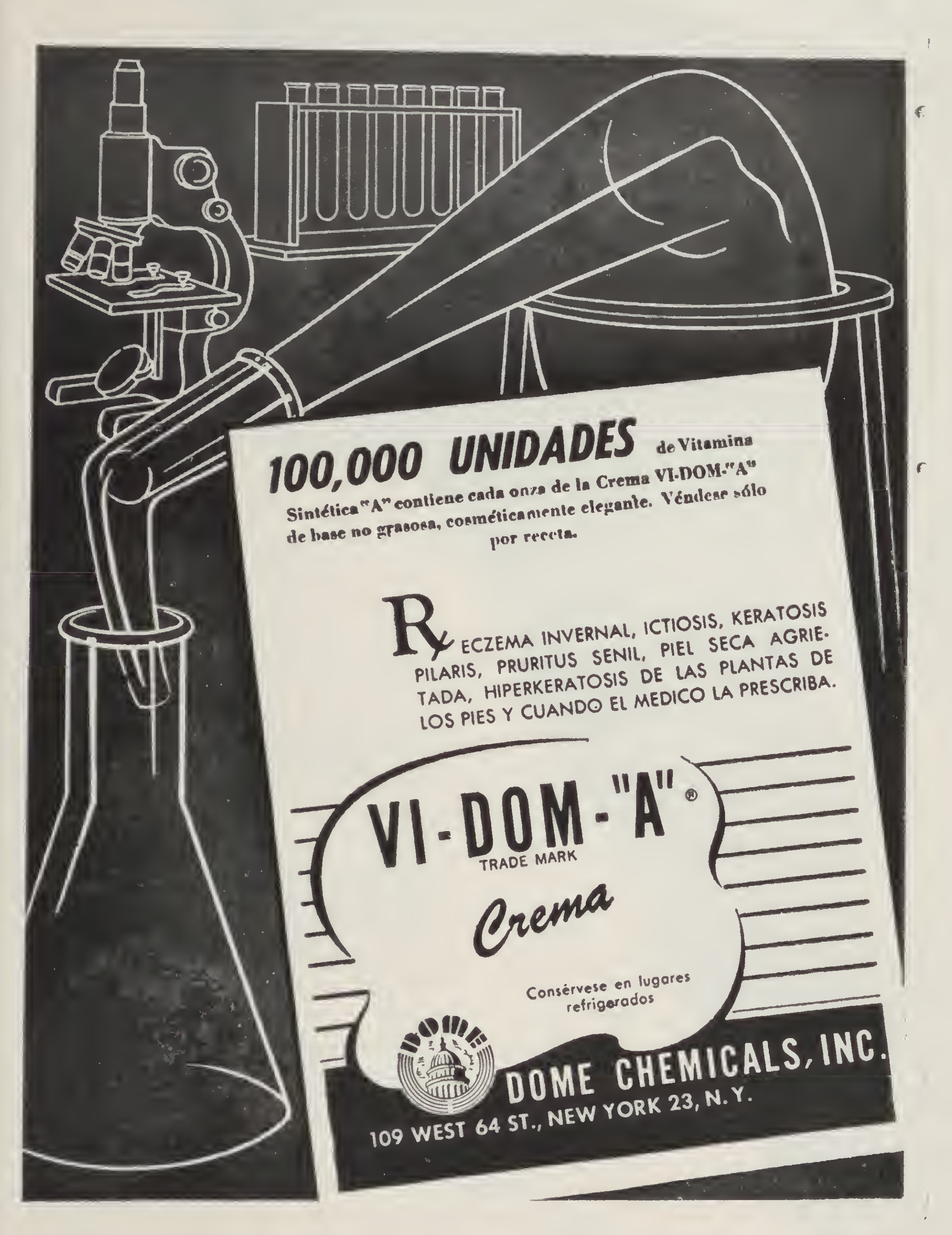


Fig. 9

(Fig. 7): "Narrowed prepylorus, may be due to spasticity; similar configuration could be produced by inflammation or even by CA. If the deformity persists after antispasmodic Rx, exploration is advised." - Third exam. (Fig. 8 & 9): "Following antispasmodic Rx again showed the narrowed prepylorus. After administration of 0.01 of Morphine, the prepyloric segment showed no abnormality."

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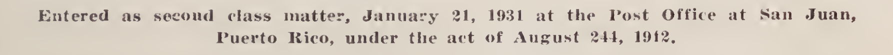
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BOLETIN

DE LA

ASOCIACION MEDICA DE PUERTO RICO

VOL. 47 JUNIO, 1955 No. 6



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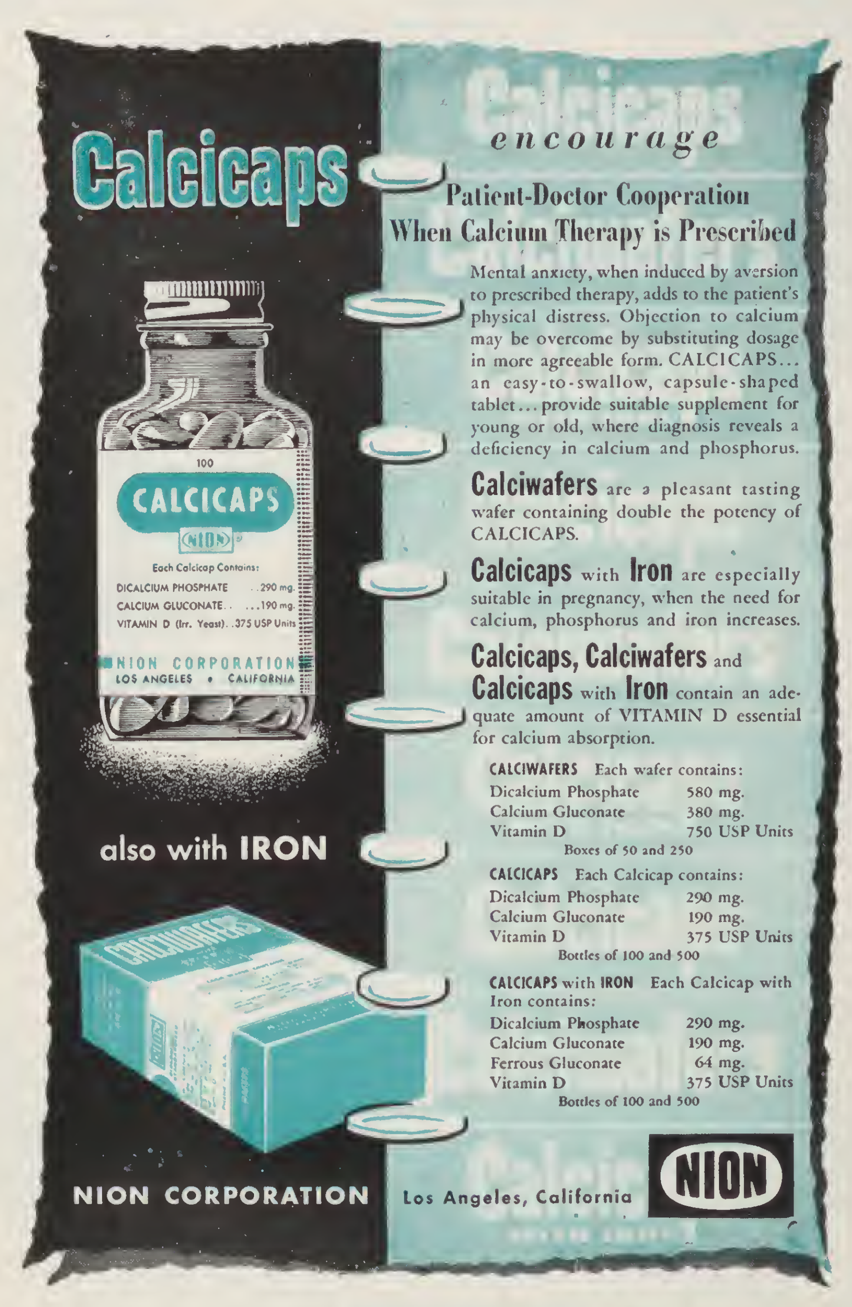
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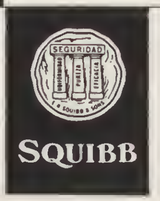
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BOLETIN

DE LA ASOCIACION MEDICA DE PUERTO RICO

VOL. 47 JUNIO, 1955 No. 6

UPPER GASTROINTESTINAL BLEEDING*

*J. A. DE JESUS, M.D., RAMON A. SIFRE, M.D. and CARLOS E. BERTRAN, M.D.***

Since the subject of gastrointestinal bleeding is so extensive we shall limit the scope of this paper to include a review of our experiences with only upper gastrointestinal bleeding in the indigent population that attends the medical wards of the San Juan City Hospital. The records of 75 patients seen during the years 1951 to 1954 inclusive were reviewed.

There were 59 male and 16 female patients in this series, a ratio of four males to one female. The age distribution is shown in Table 1. The highest incidence of bleeding occurred in the 5th decade of life.

TABLE I
AGE DISTRIBUTION

10 - 20	3
21 - 30	12
31 - 40	14
41 - 50	21
51 - 60	11
61 - 70	10
71 - 80	3
81 - 90	1
Total	75

In analyzing the various causes of bleeding several interesting points were noted. Bleeding from duodenal ulcer occurred at any age (20-88 yrs.) but was more common in the 2-3-4th decades. Bleeding from gastric cancer occurred in the 5th to the 8th decades only. Twelve of the 15 cases of bleeding varices occurred in the 5th to the 7th decades. Poisoning with suicidal intent was the cause

* Presented before the Regional Meeting of the American College of Physicians, January 29, 1955.
** From the San Juan City Hospital and the University of Puerto Rico, School of Medicine.

of bleeding in the younger age groups (17-34 yrs.). Three cases ingested HCl, one phosphorus and one lysol.

TABLE II
DISTRIBUTION OF CASES

	Cases (Ivy)	%	Cases (Ours)	%
Peptic Ulcer	3310	71.7	32	42.6
Gastric Carcinoma	478	10.3	8	10.6
Cirrhosis	201	4.4	15	20.0
Gastritis	84	1.8	2	2.6
Poison	2		5	6.3
Miscellaneous	238	6.4	2	2.6
Of undetermined cause	250	5.4	11	14.7
	4614		75	

In Table II a comparison is made of the relative frequency of upper gastrointestinal bleeding between our cases and 4614 cases collected from the literature by Ivy and his co-workers. This comparison is not intended to suggest statistical importance to our cases, but merely to point out the differences in clinical material as follows: the incidence of bleeding from peptic ulcer in our series is only 42.6% as compared with the 71.7% recorded in Ivy's study.[1] This difference is accounted for principally by the higher incidence of bleeding varices in our cases and also by the relatively large percentage of bleeding from the ingestion of chemical poisons. Among the 4,600 odd cases in Ivy's collected figures there were but 2 cases of poisoning as against 5 cases in our series.

The cause of bleeding was not established with certainty in 11 cases (14.6%). In 6 cases, complete studies failed to reveal the lesion, but for reasons beyond our control, in some of them x-ray studies were not carried out at an optimum time. Two others died soon after admission and the others left the hospital against advice or before proper studies could be completed.

The literature varies considerably regarding the incidence and mortality from hemorrhage of the upper gastro-intestinal tract.[2,3,4] This should be expected in view of the many factors concerned, one of which is the definition of the term as applied to specific cases.

Everyone has agreed regarding the meaning of occult blood as detected by chemical methods. There is no confusion regarding the meaning of manifest bleeding, which refers to the presence of red blood in the vomitus (hematemesis) or to black tarry stools (melena). A loss of 60 cc of blood will yield a dark stool

on a milk and cream diet and 80-100 cc a black stool on a general diet; even less will form a clot in the gastric contents. This is not a significant blood loss per se, yet, such a hemorrhage is sometimes referred to as "gross" or "massive" hemorrhage. In some articles, "gross" is used in the same sense as manifest or visible evidence of bleeding. On the other hand, "massive bleeding" is frequently used to indicate severe bleeding or that degree of blood loss which per se threatens life as evidenced by dizziness, weakness, fainting, or shock. Unfortunately, the appearance of these symptoms depends to a significant extent on the acuteness of hemorrhage. Hematemesis obviously indicates to some extent the acuteness and perhaps the severity regardless of the site of bleeding. It is also clear from the modern literature that hematemesis may occur without melena, even in duodenal and anastomotic ulcer, and that melena alone occurs more frequently in duodenal than gastric ulcer.

Recognizing the difficulty in classifying the cases as to the severity of bleeding we have used the following criteria:

1. The estimated amount of blood loss.
2. Presence of shock.
3. The amount of blood needed to stabilize the vital signs.
4. Hematocrit, red blood cell count and hemoglobin level.

If the estimated amount of blood loss was over 1000 cc it was considered massive bleeding. Also when the amount of blood given was 1000 cc or more. From the laboratory point of view the severity of bleeding was classified according to the criteria shown in Table III.

TABLE III

CLASSIFICATION OF BLEEDING

	Ht.	Hb.	RBC
Minimal	+40	+70	+4.0
Moderate	+25	+50	+3.0
Massive	—.25	— 50	— 3.0

TABLE IV

DEGREE OF BLEEDING

	Min.	Mod.	Mass.
Peptic Ulcer	5	12	15
Varices	0	1	14
Poisoning	3	2	0
Carcinoma	0	5	3
Gastritis	0	1	1
Hiatus Hernia	1	0	1
Undetermined	2	4	5

Using these criteria all the cases were classified as shown in Table IV. Ten cases were minimal, 24 moderate and 38 massive bleeders. While only 15 of the 32 cases of peptic ulcer bled massively; 14 out of the 15 cases of varices bled massively. Five of the eleven cases of undetermined origin bled massively.

The relative frequency of the occurrence of hematemesis and melena is shown in Table V.

TABLE V

FREQUENCY OF HEMATEMESIS & MELENA

	Hematemesis	Melena	Both
Minimal	3	2	5
Moderate	2	7	15
Massive	1	9	28*

* 3 patients passed bright red blood in the stools.

The indications for surgery in the cases that were operated are shown in Table VI. No deaths occurred in the patients bleeding from peptic ulcer disease. Only 5 of the 32 patients were operated upon, none of them for bleeding which failed to respond to medical management. An additional patient was operated upon because bleeding was not controlled by medical measures but he was not included in this report because we have been unable to locate his hospital record.

The overall mortality was 20%, and it was accounted for principally by the cases of bleeding varices. In three of the 8 deaths from bleeding varices the patients died three to ten days after the bleeding had stopped in a state of hepatic coma. It is well known that an episode of bleeding in a patient with cirrhosis may precipitate a bout of hepatic coma. One patient signed out against

TABLE VI

PEPTIC ULCER

Subtotal Gastrectomy

58 M	Gastric Ulcer
	Intractable Pain
46 F	Gastric Ulcer
59 M	Recurrent Bleeding
56 F	Intractable Pain
36 M	Previous Episodes of Bleeding (3)

medical advise while still critically ill. Another died of exsanguinating hemorrhage 13 months after the splenectomy was performed for bleeding varices. Careful follow up was not possible in the other cases.

TABLE VII
CAUSE OF BLEEDING

	Cases	Deaths
Duodenal Ulcer	28	0
Esophageal	15	8
Gastric Carcinoma	8	2
Poisoning	5	2
Gastric Ulcer	3	0
Gastritis	2	1
Hiatus Hernia	2	0
Marginal Ulcer	1	0
Undetermined	11	2
TOTAL	75	15

Among the 5 patients who bled after the ingestion of the chemical poisons HCl, lysol and phosphorus, 2 died. The one who ingested phosphorus died 2 hours after admission and the autopsy revealed bleeding from the gastric mucosa, the pancreas, and the aorta. The other patient died in his second hospitalization for ingestion of HCl after a previous unsuccessful suicidal attempt with the same poison.

SUMMARY

1. 75 cases of upper gastrointestinal bleeding have been presented.

2. The causes, the degree of severity and the mortality in our cases have been presented.

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EN TORNO AL PROGRAMA DE TUBERCULOSIS

FERNANDO PADRO, M.D.

En 1933, la tasa de mortalidad por tuberculosis alcanza la cifra de 332.5 muertes por cada 100,000 habitantes. Es la cifra más alta registrada en toda la historia de la lucha antituberculosa en Puerto Rico. El país, por entonces, estaba sufriendo de los estragos causados por los ciclones de San Felipe en 1928 y de San Ciprián en 1932. La tuberculosis campeaba por sus respetos.

Había que hacer algo. Puerto Rico no podía entregarse a las inclemencias de una enfermedad. Había que luchar. Se crean mil camas adicionales para enfermos tuberculosos. A las 400 existentes en el Hospital de Tuberculosis de Río Piedras, hoy Sanatorio Alejandro Ruiz Soler, se le añaden otras cuatrocientas. Surgen los hopitales de Tuberculosis de Guayama con 100 camas; de Ponce con 300 camas; y de Mayagüez con 200 camas. Se reorganiza el programa de control de tuberculosis.

Surgen los Centros de Tuberculosis. El problema no sólo es de diagnóstico. Hay que atacar a la enfermedad, tratando de detener su acción nefasta en el organismo humano. Estos Centros de Tuberculosis se preparan para rendir esta labor. Todos son equipados con fluoroscopios. Ahora el diagnóstico se hará con mayor certeza. Luego vendrán los equipos de Rayos-X y por último los aditamentos fotofluorográficos. Se establece un concepto nuevo en América para aquella época: La aplicación de la colapsoterapia, especialmente el neumotórax, como medida de tratamiento ambulatorio. Esta medida de salud pública, eliminando focos de contagio mediante el cierre de cavernas y la conversión de esputos positivos en negativos, aumenta indirectamente el número de camas disponibles en nuestros hospitales de tuberculosis. Se puede lograr un mejor aprovechamiento de ellas.

Las organizaciones voluntarias antituberculosas del país, que habían estado muy activas localmente ayudando y añadiendo sus esfuerzos y voluntades para la creación de hospitales de tuberculosis locales, se dieron de lleno a la nueva idea del programa de lucha antituberculosa. Allí donde faltaban fluoroscopios y otro equipo, estas Ligas antituberculosas locales lo compraron o contribuyeron en grande escala para su obtención. Las autoridades municipales no fueron menos generosas. Con esta ayuda de todos los sectores de nuestra sociedad, el programa de lucha antituberculosa pudo desarrollarse sin grandes contratiempos.

Los Centros de Tuberculosis pasaron a ser lo que son hoy en día: el principal sostén de nuestra campaña antituberculosa. Sus directores tienen que ser médicos debidamente entrenados en tu-

berculosis. No sólo conocen la parte clínica de la enfermedad, sino que a sus conocimientos en interpretación radiológica, se une un conocimiento de salud pública y de sus necesidades en relación con la tuberculosis. Las enfermeras asignadas a los Centros reciben entrenamiento especial sobre la materia. Los técnicos de Rayos-X conocen a la perfección sus obligaciones. La empleomanía de oficina está debidamente adiestrada en los diferentes menesteres que se requieren para la buena marcha administrativa de los Centros.

En estos momentos contamos con diecinueve en total. Forman parte de las Unidades de Salud Pública. La Unidad de Salud Pública que los alberga tiene que asignarle el personal necesario para su funcionamiento. Sin embargo, los Centros de Tuberculosis sirven a un número mayor de Unidades de Salud Pública con igualdad de condiciones. Los Centros de Tuberculosis son tan Centros de Tuberculosis de la Unidad de Salud Pública donde están ubicados como de las Unidades de Salud Pública dentro del área que les está asignado el servir. Los directores médicos no sólo son consultores en materias de tuberculosis, sino que actúan de moderadores y de coordinadores en estas cuestiones entre los diferentes directores de las Unidades de Salud Pública a las que sirven, actuando de freno en muchos casos debido a las limitaciones de personal y de equipo.

Los directores de las Unidades de Salud Pública son los jefes del programa de tuberculosis de su zona. Ellos preparan sus planes en coordinación con los directores de los Centros de Tuberculosis, quienes son los que conocen las limitaciones de sus equipos y personal. El Negociado de Tuberculosis formula los planes generales. Presta toda aquella ayuda que dentro de las circunstancias pueda prestar, especialmente en cuestiones de supervisión y orientación, así como en reforzar el equipo radiográfico y prestar personal técnico especializado.

Los Centros de Tuberculosis tienen a su cargo, principalmente, la búsqueda de casos de tuberculosis. Examinan a los individuos, los diagnostican y les aplican tratamiento ambulatorio. Al mismo tiempo, inician un estudio epidemiológico entre los contactos y allegados íntimos, con el fin de poder determinar el caso de origen, e investigar si hay otros casos en la vecindad. Esta restricción de búsqueda de casos está impuesta por la falta de equipo adecuado y de personal debidamente entrenado. No pueden hacerse encuestas entre grandes masas de población.

Al crearse los Centros de Tuberculosis, la búsqueda de casos se hacía a través de un examen fluoroscópico. Más tarde, los Centros fueron equipados con máquinas de Rayos-X, haciéndose desde entonces el diagnóstico definitivo con una placa grande. La fluorografía cedió paso a la fotofluorografía. Todos los Centros de Tu-

berculosis en la actualidad tienen equipos fotofluorográficos. Una fotofluorografía sospechosa no indica la presencia de la enfermedad tuberculosa. Solamente una placa grande tomada a pacientes con fotofluorografía sospechosa sirve de base para un posible diagnóstico de tuberculosis.

Toda persona mayor de 12 años es examinada gratuitamente en los Centros de Tuberculosis por medio de una fotofluorografía. A los niños menores de doce años, se les hace una fotofluorografía siempre y cuando sean referidos por un médico competente quien los ha examinado previamente y los ha encontrado con signos físicos de posible patología pulmonar. Los niños contactos de casos tuberculosos son examinados mediante prueba de tuberculina, y si positiva, se les toma una fotofluorografía o una placa grande, dependiendo de la facilidad para dicha toma.

Una fotofluorografía sospechosa demanda una placa grande. Esta última es interpretada por el director del Centro de Tuberculosis, y si positiva, el diagnóstico es dado por el médico al paciente. El record clínico se empieza. Los exámenes de contactos y allegados íntimos se llevan a cabo mediante fotofluorografías y pruebas de tuberculina. Se educa al enfermo sobre su enfermedad y lo que él puede hacer para evitar el ser un problema de salud pública. Se empieza su tratamiento.

Afortunadamente contamos hoy con las drogas antituberculosas. El neumotórax, forma original de terapia antituberculosa al fundarse los Centros de Tuberculosis, dió paso al neumoperitoneo. La colapsoterapia ha sido suplantada por las drogas como la forma principal de tratamiento activo. Sin embargo, el valor de la colapsoterapia como medida de tratamiento sigue en vigor. La estreptomicina en dosis de un gramo en inyección intramuscular cada tercer día y la isoniacida en dosis equivalentes a 4 milígramos por kilogramo de peso, en tabletas en dos tomas diarias, es el tratamiento a seguir. Cada tercer mes el resultado de este tratamiento se evalúa por medio de placas radiográficas. Puede ser ilimitado.

Un caso de tuberculosis, no importa su estado de actividad, es siempre observado periódicamente. El intervalo de los exámenes varía, pero nunca será menor de una placa anual. Casos que requieran hospitalización son enviados a los Hospitales de Tuberculosis. Aquí reciben las ventajas de un tratamiento más apropiado en una institución preparada al efecto.

Este programa de control de la tuberculosis ha estado rindiendo frutos admirables. La mortalidad por tuberculosis ha bajado a 38.3 muertes por cada 100,000 habitantes en 1954. Desde que se implantaron medidas de control adecuadas, ha habido un descenso continuo en la mortalidad, que se hizo marcadísimo durante el 1953.

Nunca en la historia de la tuberculosis en Puerto Rico se había llegado a estar por debajo de la tasa de 100 muertes por cada 100,000 habitantes. En 1952, bajó esa tasa a 93.4 muertes por cada 100,000 habitantes, y el año siguiente a 47.1.

El número de casos nuevos sigue alto. Lo que indica que la campaña de búsqueda de casos sigue dando resultados. Hay una tendencia a disminuir, pero las cifras oscilan alrededor de los 5,000 casos anuales. Fallas en el equipo radiográfico y la diferencia de sectores poblacionales examinados, pueden explicar los cambios en los números notificados.

Los Centros de Tuberculosis no atienden a todos los tuberculosos en el país. Las cifras oficiales se refieren prácticamente a los enfermos que acuden a estos Centros de Tuberculosis. Los médicos particulares están muy reacios a notificar sus casos. Como prueba de que existe más tuberculosis que lo que demuestran las cifras oficiales, está el hecho de que aproximadamente el 35% de las muertes por la enfermedad no son conocidos como casos de tuberculosis.

A pesar del progreso realizado, la tuberculosis sigue creando un problema muy serio en el campo de la salud pública. El programa de control de la enfermedad debe de mantenerse en todo su apogeo. La parte relativa a la búsqueda de casos debe de intensificarse.

Comoquiera que los Centros de Tuberculosis sirven a un área mayor que la de la Unidad de Salud Pública donde están instalados, hay que establecer un sistema coordinado entre los Centros y las Unidades adyacentes servidas por ellos para aumentar el número de personas a examinarse. Hay que facilitar el intercambio de métodos y conocimientos entre el médico del Centro de Tuberculosis y los médicos de las Unidades. Las enfermeras visitantes deben de conocer al dedillo las necesidades de los Centros para determinar la prioridad de las visitas.

Los directores de los Centros de Tuberculosis deben de visitar a todos y cada uno de los médicos de su zona, interesándolos en el problema de tuberculosis en su área y pidiéndoles su cooperación para el mejor desempeño de sus labores y una mejor notificación de casos. Debe de servirle como consultor en todos aquellos problemas de tuberculosis que le puedan ser demandados. Mayor cooperación y coordinación entre la clase médica pondría de manifiesto problemas inherentes a las condiciones locales y un plan de acción adecuado podría ser llevado a cabo.

La búsqueda de casos debe de intensificarse. Es necesario un mayor número de equipos fotofluorográficos. Sobre todo, de equipos movibles. Hay que llevar las facilidades de examen a los lugares más remotos de la isla. Para ello, es indispensable que los

oficiales médicos en salud pública, jefes de sus áreas respectivas, preparen programas al efecto. No sólo es importante saber las necesidades de un grupo poblacional específico, sino que hay que planear como afrontar esos problemas.

Las pruebas de tuberculina no han sido usadas como pruebas de diagnóstico. Su uso ha sido limitado al estudio de contactos de casos de tuberculosis menores de doce años. Aunque se han hecho ciertos estudios indirectos por motivo de la vacunación por el BCG entre otros niños, de cierto no ha habido un programa en grande escala para saber el grado de infección tuberculosa en nuestra niñez. El Negociado de Higiene Maternal e Infantil ha empezado a desarrollar un programa de investigación tuberculínica en los niños de edad pre-escolar que concurren a sus clínicas. Hay planes trazados para hacerles pruebas de tuberculina a todos los alumnos del primer grado de nuestras escuelas, anualmente. ¿Sería posible repetir la prueba a los negativos a esta prueba de tuberculina al terminar su escuela elemental? Así podríamos tener un índice de la infección tuberculosa en esta edad escolar. Actualmente, en niños de edad pre-escolar, del 15 al 20% son positivos a la prueba de tuberculina. Es muy interesante saber como se comporta el 80% restante durante sus primeros años de escuela.

La vacunación por el BCG se practicó en grande escala mediante un acuerdo entre el Departamento de Salud y el servicio de Salud Pública Federal. Luego ha sido continuada como medida preventiva en nuestros niños, sin hacer campaña al efecto. A los padres que concurren a las clínicas de las Unidades de Salud Pública, se les explica sobre la vacunación y sus ventajas. Son vacunados todos los voluntarios, niños hasta de 8 años de edad, cuya prueba a la tuberculina sea negativa. ¿Está justificada esta vacunación en nuestra población en los momentos actuales? Salvo en zonas densamente pobladas con un índice alto de morbilidad tuberculosa, donde el contagio es prácticamente imposible de evitar, no parece esta medida preventiva sea de tanta utilidad como en otros lugares. Si la isoniacida ejerce tan buena acción sobre la primo infección tuberculosa a tal extremo que evita las complicaciones más temibles, como la meningitis tuberculosa y la tuberculosis miliar, sería más factible desarrollar un programa de investigaciones tuberculínicas con miras a usar de la isoniacida si el estado de los reactores positivos a su acción lo amerita. Así se mantendría un verdadero estado investigativo, sin descansar en las posibles seguridades de una vacunación cuyos resultados no siempre son lo halagüeños que se esperan.

El tratamiento a todos y cada uno de los casos activos de tu-

berculosis debe hacerse una realidad. No debe permitirse a casos activos de la enfermedad vagar por nuestras calles, diseminando los gérmenes causales de la tuberculosis. Las drogas antituberculosas, por mejores que sean, no mejoran todas las formas de la enfermedad tuberculosa. La colapsoterapia — el neumotórax y el neumoperitoneo — no han perdido su valor terapéutico. La cirugía pulmonar y torácica se ha desarrollado grandemente. El descanso sigue siendo la base principal del tratamiento de la tuberculosis. La mejoría clínica de un paciente que llega hasta la inactividad de sus lesiones, es el producto combinado de un tratamiento juicioso dirigido por médicos especializados en la enfermedad. No todo médico tiene los conocimientos necesarios para poder llevar a cabo un tratamiento que requiere del concurso de varios métodos. El tratamiento de la tuberculosis no consiste en dar drogas solamente. Un tratamiento mal dado, incompleto, redunda en perjuicio del paciente y en un mayor problema de salud pública.

El tratamiento, a ser posible, debe ser empezado en un hospital de tuberculosis, por lo menos, hasta que las lesiones se estabilicen, para luego continuarlo ambulatoriamente. El número de camas para enfermos tuberculosos es en estos momentos de alrededor de 2,500. Es verdad que hay unas pocas camas vacías para mujeres, pero por el contrario hay una larga lista de espera para hombres. El número de camas disponible para enfermos tuberculosos es muy bajo. No debe ni siquiera pensarse en eliminar camas. Hay que aprovechar mejor las existentes. Los cinco mil casos nuevos que se descubren año tras año, debían de ser admitidos a los hospitales de tuberculosis, empezarles su tratamiento, educarlos e indoctrinarlos sobre la enfermedad, y cómo evitar el propagarla. Lesiones detenidas o inactivas que no requieran tratamiento hospitalario, pero que sí necesitan vigilancia ulterior, deben ser enviadas a los Centros de Tuberculosis para su estudio y control. Lesiones activas serían retenidas por los hospitales, hasta tanto se logre una estabilización de ellas, pasando luego a los Centros para continuar tratamiento ambulatorio. Así no habría casos de tuberculosis sin haber recibido el beneficio de un diagnóstico precoz, un tratamiento adecuado y una observación ulterior eficiente. El problema de contagio y de propagación quedaría disminuído grandemente y la salud pública en general saldría ganando. Las camas en los hospitales de tuberculosis son necesarias en grado sumo. El tratamiento quirúrgico adquiere cada día que pasa mayor importancia. Sólo en hospitales debidamente preparados para ello, se puede aplicar. Las drogas antituberculosas tienden a hacer desaparecer las lesiones inflamatorias. Las lesiones crónicas persis-

ten a pesar del mejor de los tratamientos. La cirugía elimina estos restos. Casi podríamos afirmar que puede llegarse a una curación. Este método de tratamiento requiere no sólo el equipo y los cirujanos, si no también de las camas. No puede hacerse ambulatoriamente. Hay que conservar las relativamente pocas camas que disponemos. Si posible, debemos aumentarlas.

El que el porcentaje de muertes por tuberculosis disminuya, no quiere decir que estamos acabando con la tuberculosis. Todavía nos falta mucho por hacer. Hay demasiados casos de tuberculosis sin descubrir, y de los que conocemos, hay un gran número que siguen siendo problemas de salud pública a pesar de los tratamientos impuestos. La enfermedad se adquiere mediante el contagio. No sólo es contagiosa, si no que es transmisible. Habiendo tanto caso conocido y sin conocer, la población de Puerto Rico está expuesta a contraer la enfermedad. Cada día aparecen más tuberculosos. Como la incidencia de casos es más o menos estable, y la mortalidad ha disminuído considerablemente, el número de enfermos ha aumentado. Ante esta situación ¿vamos a descontinuar parte del armamento con que contamos y que tanto nos ha servido para lograr los resultados beneficiosos que hoy estamos palpando?

El problema de búsqueda de casos sigue en pie. Tenemos que esforzarnos en tratar de conseguir el último caso de tuberculosis para ponerlo, si no bajo control en lo referente a su enfermedad, sí bajo la custodia de las autoridades competentes, cosa de tratar de evitar la propagación de la enfermedad. El aislamiento con su tratamiento correspondiente ayudaría grandemente en esta campaña. El conocer todos y cada uno de los casos de tuberculosis en Puerto Rico es imprescindible para completar un buen programa de control de la enfermedad. El examen ulterior de todos ellos es necesario para mantener la vigilancia sobre posibles recaídas y posibles aumentos de infección entre la población.

El programa de control de tuberculosis está rindiendo frutos provechosos. No por esto debemos de aflojar nuestra presión sobre la enfermedad y los problemas que acarrea. No podemos eliminar o descontinuar ni siquiera parte de lo que disponemos para combatirla y hacerle frente con el éxito con que lo hemos estado haciendo. Al contrario, para poderle asestar el golpe final, debíamos aumentar nuestros efectivos. Tenemos que mantenernos alerta contra el enemigo. Aún en su agonía nos puede causar un grave daño si nos descuidamos o nos dormimos en los laureles. En pié de lucha y cambatiendo sin cesar, no importa los sacrificios, lograremos que la tuberculosis deje de ser un problema de salud pública en Puerto Rico.

EL RIESGO DEL ACTUAL OPTIMISMO EXCESIVO EN LA EVALUACION DE LA BATALLA ANTI-TUBERCULOSA

E. FERNANDEZ CERRA, M.D.

Ante la Asamblea Anual de la Asociación General Antituberculosa de Puerto Rico celebrada, aquí en esta Asociación, aproximadamente un mes, pronuncié estas palabras dando voz de alarma sobre el riesgo de la injustificada reducción en el número de camas disponibles para la hospitalización y tratamiento de los casos tuberculosos en Puerto Rico que contempla nuestro Departamento de Salud.

A invitación de los organizadores de este panel, que creyeron que mis palabras de alarma debieran tener mayor difusión, repetiré en parte la conferencia de esa noche.

Quiero sin embargo, decir que mi posición es clara; es la de un tisiólogo, vivamente interesado por los problemas de nuestro país, por afición y por herencia, y en este caso, muy específicamente en los problemas de salud.

No me anima más interés que la preocupación motivada por las frecuentes y metódicas manifestaciones que, en boca de las autoridades pertinentes, han visto la luz recientemente en la prensa de nuestro país, propalando una contemplada reducción mayormente en las facilidades para el tratamiento de la tuberculosis en Puerto Rico. Manifestaciones que entiendo tienen y seguirán teniendo, de no ser modificadas, el nocivo efecto de crear un falso sentido de seguridad y una serie de confusiones y dudas entre el público en general, los pacientes y sus familiares, al inferirse de ellas, lógicamente, que la tuberculosis está erradicada en Puerto Rico y ha dejado de ser un problema de salud pública, o que ya no es necesario hospitalizar, o no recibirán mayores beneficios aquellos pacientes que padeciendo de tuberculosis fuesen hospitalizados durante la fase activa de su enfermedad.

Me propongo desvirtuar ambas inferencias.

Todos coincidimos en que la declinación de la mortalidad por tuberculosis, iniciada hace muchos años en los países civilizados, se ha acelerado considerablemente desde que se introdujeron los tuberculostáticos en el tratamiento de la enfermedad. Sin embargo, todos también coincidimos en que la morbilidad no ha seguido el mismo camino o sea no ha ocurrido una declinación correspondiente. El número de casos nuevos disminuye muy poco y como resultado ha habido un aumento considerable en el sector tuberculoso de la población. O sea, hoy hay mayor número de tuberculosos que hace dos años.

Es un hecho bien conocido que la tasa de mortalidad de una

enfermedad determinada no puede utilizarse como índice de frecuencia o como base para el planeamiento de medidas de control de la misma. Aunque la tuberculosis no es una excepción a la regla, hasta ahora y durante muchos años, los cálculos sobre su prevalencia y sobre sus medidas de control se han basado en las cifras de mortalidad. Se consideraba que por cada muerte por tuberculosis había 10 casos de la misma en la población y que se deberían de proveer para los mismos 2.5 camas por cada muerte, o sea por cada 10 casos de tuberculosis en la población.

Desde que se introdujeron los antibióticos en 1946 y sobre todo desde el uso del INH en 1952, debido a la discrepancia tan marcada y significativa entre el curso de la mortalidad y morbilidad, todos los cálculos basados sobre mortalidad han perdido su validez. Continuar aferrados a este sistema conducirá a tremendos errores puesto que el dramático descenso en la mortalidad sólo ha conseguido representar en forma falsa el problema de la tuberculosis. Para mejor comprender la evolución de la enfermedad y tener un cuadro más real de la misma es preciso estudiar más a fondo la morbilidad. O sea que lo que importa saber es el número de enfermos que hay, ya que los índices obituarios no sirven para darnos esa información. Desgraciadamente, la información de morbilidad que existe es poco confiable ya que hasta recientemente se dependía de la mortalidad para hacer presunciones sobre morbilidad.

Para demostrar la importancia de tener conocimiento sobre la morbilidad de la enfermedad y de refrenarnos de continuar haciendo cálculos a base de mortalidad voy a utilizar las últimas cifras disponibles sobre Puerto Rico para probar este punto:

En Puerto Rico, el índice de mortalidad en 1950 era de 129.6 por 100,000 habitantes y murieron 2,861 personas. Utilizando el cálculo, válido entonces, de 10 casos de tuberculosis por cada muerte encontramos que había entonces aproximadamente 28,610 casos de tuberculosis en Puerto Rico ese año. Esta cifra no está equivocada pues según estudios de la Unión Internacional Contra La Tuberculosis se considera que en países de mortalidad alta, y Puerto Rico en 1950 era uno de ellos, más del 1% de la población tiene tuberculosis. Si aplicamos este dato a la población de Puerto Rico a esa fecha notamos que la cifra que se obtiene es compatible con la de los 28,000 casos calculados por el índice de mortalidad.

Según cifras oficiales murieron por tuberculosis

en	1951	----	2,654 personas
	1952	----	2,092 "
	1953	----	1,046 "
	1954	----	alrededor de 850 personas (no te-

nemos cifras exactas, pero sabemos que el índice de mortalidad para ese año fué de 38 por 100,000 habitantes.)

Total ____ 6,592

De manera, que de los 28,610 tuberculosos que había en 1950 fallecieron aproximadamente 6,592 hasta el 1954, quedando entonces alrededor de 22,000. Sin embargo, desde 1950 para acá se han estado descubriendo casos nuevos anualmente, alrededor de 5,000 por año, los cuales tenemos que añadir a los 22,000 que quedaban, obteniendo así un total aproximado de 40,000 casos de tuberculosis para el año 1954. Así que con un índice de mortalidad de 38 por 100,000 habitantes había 40,000 casos en 1954, cuando en 1950 con un índice de mortalidad más alto, de 129.6 por 100,000, había sólo 28,000 casos. Por esta discrepancia entre mortalidad y morbilidad, antes mencionada, hemos creado un mayor número de casos de tuberculosis en la población o sea un sobrante en términos de contabilidad.

El problema es saber qué número de esos 40,000 casos están activos y son focos de propagación de la enfermedad. Como no existen cifras en ese sentido tenemos que llegar a las siguientes conclusiones:

1. Los nuevos tuberculostáticos en el tratamiento de la tuberculosis producen una sensación de bienestar marcada en los pacientes. En aquellos que tienen síntomas son librados de ellos con relativa rapidez. Se cura un número substancial de pacientes y también se cronifican o vuelven crónicos un número también substancial de pacientes que normalmente hubiesen muerto, prolongándoles la vida permitiéndoles ser focos de infección por varios años más. Los que tratamos tuberculosis hemos visto moribundos, deshauciados, perdidas todas las esperanzas, que literalmente como Lázaro se han levantado y andado. Muchos de esos pacientes hoy en día están vivos, pero no curados y debido a la sensación de bienestar que sienten, la mayoría de ellos no están hospitalizados.

2. El número de recaídas o recidivas es mayor que antes del advenimiento de las drogas. Esto se debe a varias razones —primero— estas drogas son meramente bacteriostáticas y no bactericidas, o sea no matan el bacilo que causa la tuberculosis; meramente impiden su reproducción.

Segundo—los pacientes tienden a abandonar el tratamiento en mayores números que antes sin estar el mismo concluído. Esto se debe principalmente a la sensación de bienestar que obtienen y al falso sentido de seguridad que la prensa laica ha contribuído a crear entre muchos de ellos debido a manifestaciones exageradamente optimistas o poco exactas.

En tercer lugar hay un número substancial de pacientes, po-

siblemente en mayores cantidades en comunidades pequeñas, que están siendo tratados por médicos generales cuyos conocimientos de la patogenia de la enfermedad son escasos o limitados. Esto es principalmente debido a que por generaciones el estigma de ser conocido como tuberculoso ha sido tal que a muchas personas no les gusta les vean entrar en oficinas de especialistas en esta enfermedad y por lo tanto prefieren ir a oficinas de médicos generales a que le den el tratamiento de inyecciones y píldoras para dicha enfermedad. Anteriormente, debido a lo altamente especializado y técnico del tratamiento de la tuberculosis un enfermo de la misma se veía obligado a recurrir ya al especilista privado, ya al centro de salud a obtener tratamiento para la misma. Sin embargo, como hoy en día se conoce generalmente el tratamiento quimioterapéutico de la enfermedad y se saben generalmente las dosis a aplicarse, le es fácil al médico general recetar el mismo. Desde el punto de vista de salud pública ésto tiene, además, la implicación de que estos casos no son reportados al Negociado de Tuberculosis y por lo tanto no se obtienen cifras verdaderas en cuanto a la morbilidad.

Cuarto — Desarrollo de resistencia de parte del bacilo de la tuberculosis a los bacteriostáticos. El bacilo de la tuberculosis tiene la facultad de poder desarrollar resistencia o defensas contra estas drogas. Esta facultad aumenta según progresa el tratamiento. De manera, que todo caso mal tratado tiene la desventaja de posiblemente las drogas haber perdido su efectividad debido al desarrollo de resistencia a las mismas y por lo tanto el tratamiento futuro ser mucho más difícil y complicado.

Si todas estas razones no fueran suficientes para hacer reconsiderar cualquier reducción en el número de camas se me ocurren que todavía no hemos discutido dos datos que son de singular importancia: primero — el reposo es insustituíble e imprescindible en el tratamiento de la tuberculosis. Ningún tratamiento pasado o presente substituye al reposo en el tratamiento de esta enfermedad. Esta es la base sólida sobre la cual se echan los cimientos de la curación. Hemos visto que aquellos pacientes, que en adición a otros tratamientos, observan un reposo adecuado no sólo obtienen mejores resultados en sus curaciones sino que los obtienen más rápidamente que aquellos que no observan reposo.

Segundo — Desde el punto de vista de salud pública todavía sigue siendo el mejor medio de evitar el contagio el aislar a los casos activos.

Los tratamientos ambulatorios para tuberculosis solamente están justificados cuando hay escasez de camas o cuando el paciente está imposibilitado por alguna razón muy importante de hospitalizarse durante la fase activa de su enfermedad. Es un dato bien

establecido que países que mantienen una proporción alta de camas por paciente, tienen mortalidad más baja que aquellos donde no es así. La historia de la lucha antituberculosa en Puerto Rico creo nos ofrece una comprobación de ésto: En el 1932 se estableció en Puerto Rico el Negociado de Tuberculosis del Departamento de Salud. Entonces la mortalidad era de 205 por 100,000 habitantes. Este índice de mortalidad subió a 332 por 100,000 en 1933 a pesar de todos los esfuerzos de dicho negociado. En 1933 se añaden 800 camas y los dispensarios del Departamento de Salud se convierten en centros y a pesar de Puerto Rico estar atravesando por una depresión económica el índice de mortalidad bajó a 207 por 100,000 habitantes en 1946. Luego, al añadirse la Estreptomicina y el Parasal como ayudas adicionales bajó dicho índice de mortalidad a 93.4 por 100,000 en 1952.

En 1952 ocurrieron dos hechos fortuitos en la historia de la lucha antituberculosa en este país — primero, el uso de la Isoniacida como arma valiosa en el tratamiento de la tuberculosis y segundo se añaden 800 camas más a las que ya había disponibles para la hospitalización de enfermos tuberculosos. Declina entonces la mortalidad a 38 por 100,000 en 1954. Sin embargo, entiendo que esta reducción tan dramática y tan halagadora no se debe exclusivamente al uso de la Isoniacida sino también en una parte considerable al incremento en el número de camas.

Debido a la ausencia de otras razones nos vemos obligados a considerar que ha sido la dramática disminución en mortalidad lo que ha movido a considerar una reducción en el número de camas.

Creo haber demostrado que de continuar utilizando ese criterio nos conducirá a lamentables errores. Para propósitos de discusión utilizaremos las cifras de mortalidad para con ellas demostrar que aún con la mortalidad y morbilidad que tenemos ahora en Puerto Rico no es justificable, bajo consideración alguna, el desintensificar la lucha antituberculosa.

En el 1950 el índice de mortalidad por tuberculosis en Puerto Rico era 129.6 por 100,000 habitantes. Sin embargo, entonces había 31 países con mortalidad más baja que la de Puerto Rico. Y de éstos 16 tenían una mortalidad menor que la que tiene Puerto Rico hoy día y a pesar de tener una mayor proporción de camas por pacientes que Puerto Rico, ninguno consideró reducir el número de las mismas.

En 1952 el índice de mortalidad de Puerto Rico era de 93.4. Países como Escocia con 31.3, Suiza (24.8), Inglaterra (24.1), Canada (17), Estados Unidos (16.1) y Dinamarca (8), todas cifras menores que las actuales en Puerto Rico y vuelvo y repito, mayor proporción de camas por pacientes que las que existen en Puerto Rico, ninguno consideró reducir el número de camas ni convertir

sus sanatorios en hospitales generales, no importa cual fuese la necesidad de los mismos.

En 1953 Estados Unidos con un índice de mortalidad de 12.3 y de morbilidad de 52.6 por 100,000 habitantes (Puerto Rico tiene ahora mortalidad de 38 y morbilidad de 207 por 100,000 en 1953), no se consideró reducción alguna. Los sanatorios que se han cerrado se ha debido principalmente a la localización de los mismos. Estos fueron principalmente construídos en la época en que el tratamiento de la tuberculosis era exclusivamente climático y por lo tanto estaban localizados en lugares retirados y remotos. Como hoy en día se puede proveer el mismo tratamiento en las grandes ciudades, los pacientes prefieren obtenerlos cerca y no acudir a estos sanatorios lejanos.

Entiendo que el porvenir de la lucha antituberculosa depende del balance final entre los casos curados y el número de casos crónicos. De aplicarse juiciosamente los tratamientos, haciendo uso de todas las facilidades disponibles para así aumentar el número de curados y reducir el de crónicos, y de aislarse los casos activos para evitar propagación de la enfermedad, es lógico pensar que llegará el momento en que el balance sea favorable.

Procede, pues, hacer propaganda en el sentido de educar a la población a valerse de todas las facilidades asistenciales que estén indicadas en cada caso y además condenar el uso indiscriminado de bacteriostáticos, para garantizar así mayor número de curaciones. Hay que desvanecer el error, entre los profanos, que la enfermedad está vencida. Hay que imbuir en el espíritu del público la idea de que los bacteriostáticos no pueden reemplazar, por si solos, al tratamiento clásico de la enfermedad y que para obtener de la medicación el máximo rendimiento ha de integrársela a un plan coordinado en el cual figuren también el reposo, la colapsoterapia y en determinados casos la cirugía de exéresis, si queremos que la tuberculosis deje de ser un problema en pocos años.

Debemos evitar crear, con expresiones exageradamente optimistas, ilusiones terapéuticas y un falso sentido de seguridad entre los enfermos y sus familiares, puesto que los perjuicios de un tratamiento mal llevado son con frecuencia irreparables para ellos y muy bien podrían serlo para Puerto Rico.

TUBERCULOSIS EN LOS NIÑOS



*JOSE E. SIFONTES, M.D.**

Desde el punto de vista del Pediatra, el futuro de la tuberculosis en el niño puertorriqueño depende de las características de la enfermedad según se observa en nuestra población infantil.

Nuestros niños adquieren la enfermedad casi siempre mediante contacto con un adulto tuberculoso. Este es capaz de transmitir la enfermedad al niño, no solamente antes de que se haya descubierto y tratado la enfermedad en el adulto sino, que en muchos casos después de haber iniciado tratamiento, con las drogas y a veces, aún, después de haberlo completado.

Al adquirir la enfermedad, el niño desarrolla una infección primaria pulmonar. Poco tiempo después viene una diseminación hematógena casi siempre, oculta, pero capaz de producir la tuberculosis extra-pulmonar comunmente observada en el niño, como tuberculosis ósea y meningitis tuberculosa.

En la mayoría de los casos la lesión pulmonar primaria en el niño pasa inadvertida y se cura, dejando como huella, algunas calcificaciones o un pequeño foco invisible de tuberculosis en los ápices pulmonares.

Varios años o décadas, más tarde, durante un período de crisis en la vida del individuo, este foco apical puede reactivarse y producir la tuberculosis de tipo reinfección que se observa en el adulto. Es muy probable que la gran mayoría de los adultos tuberculosos adquirieron la enfermedad durante la niñez.

El futuro de la tuberculosis en nuestros niños y quizás en nuestros adultos dependerá principalmente del grado de contagio que está recibiendo nuestra población infantil actualmente de los adultos tuberculosos.

Puede estudiarse esta situación, a través de estudios de tuberculina en los niños menores de 3 años de edad, o sea aquellos que han nacido luego de haberse descubierto la isoniacida.

En mis estudios, he podido observar, áreas urbanas donde solamente el dos por ciento de estos niños, o sea menos de tres años de edad, han tenido su prima infección tuberculosa. Mientras que tenemos arrabales donde 52% de los niños menores de tres años de edad ya han tenido tuberculosis primaria. En estos arrabales se sigue incubando una gran fuente de tuberculosis que podría perpetuar la enfermedad en nuestra población y que actualmente está enviando el mayor número de niños menores de 3 años con tuberculosis a ingreso a nuestro Sanatorio. Por esta razón el propósito de nuestro trabajo de investigación es el control de la Tuberculosis Primaria y sus complicaciones, tales como la meningitis tu-

berculosa, tuberculosis ósea y la Tuberculosis Pulmonar progresiva.

Estamos usando la isoniacida en niños con Primainfección Tuberculosa para determinar si será posible, evitar estas complicaciones, curando la enfermedad en sus principios.

Aún no sabemos, cuales serán los resultados de esta investigación y pasarán varios años ante de que podamos llegar a conclusiones finales.

Mientras tanto el mejor medio de controlar la tuberculosis en el niño es evitando el contacto del adulto tuberculoso con los niños. Cuando esto es inevitable, podría vacunarse estos niños con BCG, entendiéndose que aún deben usarse precauciones, ya que la BCG no ofrece protección absoluta.

De acuerdo con mis observaciones personales, vislumbro este cuadro ideal para el control de la Tuberculosis en el niño: se evitaría el contagio de los niños mediante una campaña de educación al público que permitiera el diagnóstico, tratamiento y hospitalización del adulto tuberculoso en las fases mínimas de la enfermedad. Los enfermos persistentemente positivos debieran permanecer aislados y a la misma vez, recibir instrucción especial del peligro que ellos representan para sus semejantes.

Por otra parte se harían pruebas de Tuberculina cada seis meses en todos los niños sanos de manera, que tan pronto el viraje tuberculínico, indicara la infección primaria, se iniciara el tratamiento con isoniacida u otra droga nueva, que permitiera curar la enfermedad antes de que esta se desarrollara. Así no veríamos más niños lisiados permanentemente por la tuberculosis ósea, meningitis tuberculosa y lesiones avanzadas pulmonares.

PAGINA DEL PRESIDENTE



Programa Radial

Cumpliendo con otro de nuestros objetivos, el domingo 19 de junio iniciamos una serie de programas radiales con la idea de intensificar nuestra labor de relaciones públicas, llevando a la comunidad información genuina sobre la labor que realiza la clase médica en el país y sobre los adelantos alcanzados por la ciencia médica.

La transmisión de este programa es posible gracias a la cooperación generosa que nos ha dado la Madison Pharmaceutical Laboratories, Inc. y a la colaboración que nos han prometido los compañeros a quienes nos hemos acercado.

En los dos primeros programas del mes hemos tenido la ayuda valiosa del honorable Secretario de Salud, doctor Juan A. Pons, quien ha discutido en términos muy apropiados todo lo concerniente al problema de la poliomielitis y la vacuna Salk.

Cordialmente invitamos a todos los compañeros a escuchar este programa todos los domingos a las doce del mediodía, a través de la WKAQ o las radioemisoras de Ponce y Mayagüez que retrasmiten el mismo y que exhorten a sus familiares y amigos a que nos honren con su atención. Quedan igualmente todos invitados para colaborar en el programa. Aquellos que deseen brindarnos ayuda pueden dirigirse a la secretaría de nuestra Asociación o al Comité de Relaciones Públicas.

Boletín Médico

Con la presente edición del Boletín, la Junta Editora ha logrado poner al día la publicación de nuestra revista.

El esfuerzo realizado por los compañeros editores es merecedor de nuestro sincero reconocimiento y para que puedan seguir llevando a cabo su tarea con la diligencia hasta ahora demostrada, es preciso que todos los miembros de la Asociación les brindemos la mayor cooperación posible.

Sabemos que muchos compañeros tienen sobre sus escritorios artículos pendientes de varios toques finales para su inmediata publicación. Cordialmente les invitamos a dedicar a esa tarea algunas horas, de manera que puedan remitir su artículo a la Junta Editora en el curso de los próximos días.

Servicio Médico Central

La Asociación Médica del Distrito Este sigue operando con bastante éxito el plan de servicio médico, mediante el cual se gestionan servicios médicos para todas las personas que así lo soliciten en el área metropolitana a cualquier hora durante todos los días del año.

Hemos notado sin embargo, que el número de médicos generales dispuestos a atender cualquier llamada, ha venido disminuyendo año tras año, y esto puede ser un obstáculo serio en el desenvolvimiento del programa.

Deseamos aprovechar esta ocasión para instar a los jóvenes médicos residentes en el área metropolitana a que se suscriban al plan de "Servicio Médico Central" y cooperen con la Asociación Médica del Distrito Este en el sostenimiento del mismo, para beneficio de la comunidad en general.

Asociación Médica Americana

Recientemente asistimos a la asamblea anual de la Asociación Méd'ca Americana celebrada la primera semana del mes en curso en Atlantic City y tuvimos el honor de conocer y hablar un rato con su nuevo presidente, el doctor Elmer Hess, quien nos ratificó su intención de asistir a nuestra asamblea anual en diciembre. Concurrimos a varios de los actos celebrados y permanecimos toda una tarde observando una de las sesiones celebradas por la Cámara de Delegados de dicha agrupación.

La labor que realiza la Asociación Médica Americana en defensa de los derechos del médico y por el bien de la comunidad, es una sin paralelo en el mundo, y por ello es acreedora a que todos los médicos le brindemos nuestra más ferviente ayuda. Pertenecer a la gran familia de médicos americanos es un deber a la par que un honor para todo médico.

Aquellos que aún no son miembros de la A.M.A. tienen ahora la oportunidad de hacerlo pagando solamente la cuota de medio año, o sea $12.50. Haga su cheque a nombre de "American Medical Association" y remítalo por conducto de nuestra Asociación.

Legislación

En otra sección de este Boletín publicamos el Proyecto sustituto del Senado #511, cuyo propósito es eliminar las Juntas Examinadoras, entre éstas la Junta Examinadora de Médicos.

Esta medida legislativa será objeto de vistas públicas en la próxima sesión de la Legislatura Estatal, y en dicha ocasión de-

bemos todos presentarnos a luchar por la existencia de nuestro Tribunal Médico, para garantizar al pueblo puertorriqueño el alto nivel médico alcanzado a través de muchos años de luchas y sacrificios.

Recomendamos a todos los compañeros estudiar detenidamente el mencionado proyecto y a prepararse para presentar un frente unido en defensa de nuestros principios.

Dr. Ricardo F. Fernández
Presidente

NOTAS NECROLOGICAS

Dr. Pedro Conde: El viernes, 3 de junio de 1955, falleció en Santurce, víctima de un derrame cerebral, el compañero cuyo nombre encabeza estas líneas.

El doctor Conde nació en San Juan, el día 12 de abril de 1898, y durante sus años mozos ejerció como Ministro de la Iglesia Bautista en la ciudad de Caguas. Luego estudió medicina en la Universidad de La Habana, graduándose de Doctor en Medicina en agosto de 1943. Hizo su internado en el Hospital de Distrito de Arecibo y fué admitido a ejercer en Puerto Rico el día 26 de abril de 1946. Fué médco de beneficencia en Arecibo y Sabana Grande, y más tarde se trasladó a Santurce, donde ejercía como médico privado al mismo tiempo que ocupaba un cargo en el Servicio de dispensario del Hospital de la Capital, durante el desempeño del cual le sobrevino el derrame cerebral que le privó de la vida.

El compañero fenecido formó parte de la Asociación Médica desde el 8 de octubre de 1947 y era un asíduo concurrente a los actos científicos auspiciados por nuestra agrupación.

Sobreviven al amigo fenecido, su esposa la señora Dolores Larrosa y su hermano, Joaquín Conde, a quienes deseamos reiterar nuestra sentida expresión de condolencia.

✧ ❖ ✧

Dr. Federico Valázquez: En la mañana del sábado 18 de junio falleció en su rensidencia en Santurce, víctima de una trombosis coronaria, este querido compañero.

El doctor Velázquez nació en Santo Domingo en el año 1904; se recibió de Doctor en Medicina en la Universidad de París en

julio de 1934, e ingresó en nuestra Asociación el 27 de octubre de 1937. Desde que llegó a nuestra Isla estuvo conectado con el Departamento de Salud, primero como médico del Sanatorio insular (Sanatorio Alejandro Ruiz Soler) y más tarde como médico del Centro Antituberculoso de la Unidad de Salud Pública de Santurce.

También tenía el doctor Velázquez su propia oficina donde se dedicaba al tratamiento de enfermedades del pecho.

En el 1943 el doctor Velázquez fué el ganador del Premio al mejor trabajo médico realizado en nuestra Isla, creado por la Legislatura insular a instancias del doctor Manuel de la Pila Iglesias, por su aportación **"Un nuevo método para el estudio de la cinemática de la sombra cardiovascular"**, y mediante el cual el compañero fenecido, dió nuevas pruebas de su genio inventivo, que ya en años anteriores durante su época de estudiante lo había conducido a desarrollar varios inventos que fueron aceptados y registrados por la Oficina de Patentes en Washington.

Sobreviven al compañero fenecido su esposa, señora Marie Louise Alrivy, y sus hermanos, Rafael, Guaroa, Caridad y Luz, a quienes reiteramos nuestra más sentida condolencia.

✧ ❖ ✧

En nuestra próxima edición publicaremos una Nota Necrológica sobre el doctor Federico Velázquez que nos envió el doctor Koppisch cuando ya estaba en prensa este número del Boletín.

O

INFORME DETALLADO DEL DESENVOLVIMIENTO DEL AUXILIO MEDICO MUTUO DESDE LA FECHA DE SU FUNDACION (1943) HASTA EL DIA 15 DE JUNIO DE 1955

Plácenos transcribir a continuación para conocimiento de la matrícula un resumen del funcionamiento del Auxilio Médico Mutuo, desde la fecha de su organización (noviembre 1942) hasta el 15 de junio de 1955.

Número actual de miembros de la Asociación ___ 692
(Del total de la matrícula hay 54 en los Estados Unidos y en las fuerzas armadas).

De la matrícula actual hay 466 que están entre los 25 y 50 años de edad. Los restantes 226 están entre los 51 y 89 años de edad.

Durante los 12 años de funcionamiento el Auxilio ha pagado 50 pólizas con un total de ________ $62,267.58
(Una de estas pólizas fué pagada en vida del médico)

De acuerdo con esto el promedio anual de muertes ha sido **cuatro.**

Al 15 de junio de 1955 el Auxilio Médico Mutuo tiene un capital de ________________________ $57,769.96

✧ ❖ ✧

Las siguientes tablas son demostrativas de la edad de los compañeros que integran actualmente nuestra Asociación, así como una relación de las pólizas pagadas por el Auxilio hasta la fecha.

DISTRIBUCION DE LA MATRICULA DE LA ASOCIACION POR GRUPO DE EDADES

Edad	Número	Por ciento
25 - 30	53	7.8
31 - 40	249	36.3
41 - 50	164	23.6
51 - 60	133	19.4
61 - 70	76	10.9
71 - 80	15	1.8
81 - 90	2	.2
	692	

DISTRIBUCION DE SOCIOS DEL AUXILIO POR EDAD — AL 15 DE JUNIO, 1955

Edad	Núm.	Edad	Núm.	Edad	Núm.
25	1	46	16	67	6
26	2	47	12	68	3
27	10	48	18	69	3
28	10	49	18	70	3
29	16	50	12	71	2
30	14	51	12	72	3
31	20	52	10	73	1
32	21	53	12	74	2
33	30	54	10	75	1
34	16	55	18	76	1
35	24	56	18	77	0
36	36	57	9	78	2
37	26	58	16	79	1
38	35	59	10	80	2
39	24	60	18	81	0
40	17	61	9	82	1
41	15	62	16	83	0
42	13	63	10	84	0
43	16	64	6	85	0
44	18	65	10	86	0
45	26	66	10	89	1
			Total		692

DISTRIBUCION DE MEDICOS FENECIDOS POR EDAD

Edad	Numero médicos fenecidos
25 - 30	1
31 - 40	2
41 - 50	8
51 - 60	20
61 - 70	12
71 - 80	4
81 - 90	2
Total	49

POLIZAS PAGADAS HASTA LA FECHA — JUNIO 15, 1955

Nombre médico fenecido	Edad a su fallecimiento	Importe pagado a los beneficiarios
1943		
1. R. López Nussa	57	$1,000.00
1944		
2. A. Villenueve	58	1,000.00
3. José E. Igartúa	54	1,000.00
4. Francisco Seín	75	1,009.31
5. Jaime Vilá Morales	44	1,009.31
6. José Belén Gotay	49	1,009.31
7. M. Riera López	48	1,009.31
1945		
8. José M. Santiago	65	1,009.31
9. Jaime Rivas Soto	56	1,009.31
10. Miguel Roses Artau	70	1,009.31
11. Charis Gould	40	1,009.31
12. Félix Alfaro Díaz	53	1,009.31
13. Julio Cintrón Motta	69	1,009.31
1946		
14. E. Fernández García	57	1,009.31
15. Luis Falagán	41	1,009.31
16. M. Cortés Quintana	52	1,009.31
17. J. Martínez Rodríguez	54	1,009.31
1947		
18. Ramón Berríos	52	1,009.31
19. M. García de Quevedo	57	1,009.31
20. Levis C. Babcock	75	1,009.31
21. Walter A. Glines	66	1,009.31
1948		
22. Rafael V. Lange	66	1,020.00
23. Agustín Mújica	49	1,020.00
1949		
24. Alexander T. Cooper	66	1,020.00
25. A. Romeu Ortiz	52	1,020.00
26. Vicente Roure	83	1,020.00

Nombre médico fenecido	Edad a su fallecimiento	Importe pagado a los beneficiarios
1950		
27. Guillermo Salazar	69	1,500.00
28. Jorge del Toro	66	1,500.00
29. Laureano Trelles	54	1,500.00
30. Jaime Costas Díaz	61	1,500.00
31. E. J. Cabán	57	1,500.00
32. Manuel de la Pila	66	1,500.00
33. J. L. Montalvo Guenard	65	1,500.00
1951		
34. J. Cerra Quiñones	30	1,500.00
35. José R. Maymí	48	1,500.00
36. Carlos Encarnación	50	1,500.00
37. J. R. Lanauze Rolón	59	1,500.00
38. Pedro J. Palou	(Pagada en vida)	1,500.00
1952		
39. José Añeses	37	1,500.00
1953		
40. E. Garrido Morales	53	1,500.00
41. Arturo Torregrosa	70	1,500.00
42. Ramón Lavandero	66	1,500.00
1954		
43. Amalio Roldán	74	1,500.00
44. Domingo Crescioni	64	1,500.00
45. I. González Martínez	83	1,500.00
46. José N. Gándara	47	1,500.00
47. Francisco M. Susoni	77	1,500.00
1955		
48. Hilario Caso	64	1,500.00
49. Alvaro Santaella	67	1,500.00
50. Pedro Conde	57	1,500.00
	Total	$62,267.58

SECCION DE LA BIBLIOTECA

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Servicio Bibliotecario

Deseamos recordar a la matrícula que la Biblioteca de la Asociación permanece abierta todas las noches, de 8:00 a 10:00, y los sábados de 9:00 a 12:00 de la mañana, excepto los días feriados.

No obstante, cualquier miembro que prefiera hacer uso de la Biblioteca durante las horas laborables del día, gustosamente será atendido por alguno de los empleados de la Asociación.

Revistas Médicas

En la próxima edición del Boletín publicaremos una relación de todas las revistas médicas que regularmente recibimos en la Biblioteca.

Revise dicha lista y si Ud. desea obsequiarnos una suscripción a alguna otra le estaremos muy agradecidos.

Cordialmente le exhortamos a utilizar nuestro Servicio de Biblioteca.

PROYECTO DEL SENADO 511 PARA ELIMINAR LAS JUNTAS EXAMINADORAS

Para conocimiento general de nuestros lectores, nos complacemos en transcribir a continuación el Sustituto al Proyecto del Senado 511, cuyo propósito es eliminar las Juntas Examinadoras, entre éstas la Junta Examinadora de Médicos.

Dicho proyecto fué presentado en la pasada sesión legislativa y será objeto de discusión en la próxima. Suplicamos a los médicos lean cuidadosamente esta legislación, de manera que puedan ayudar a nuestros dirigentes a combatirlo cuando se celebren vistas públicas en relación con el mismo.

SUSTITUTO AL P. DEL S. 511

9 de mayo de 1955

Presentado por Comisión Especial del Senado

L E Y

Para transferir a los Secretarios de ciertos departamentos gubernamentales del Estado Libre Asociado de Puerto Rico y al Jefe del Servicio de Bomberos de Puerto Rico, todas las funciones, facultades, poderes, derechos, deberes, archivos, documentos y fondos de las Juntas y Tribunales Examinadores organizados al amparo de diversas leyes de la Asamblea Legislativa, al igual que las facultades, poderes y deberes que hasta ahora han desempeñado los presidentes y miembros de dichos organismos al amparo y de acuerdo con las leyes básicas que regulan dichos organismos, según enmendadas, las cuales quedan en vigor como Reglamentos básicos del Secretario, así como para transferir a dichos Secretarios el personal de la "Oficina Administrativa de Juntas Examinadoras y Juntas de Registro"; para facultar a los Secretarios a delegar en funcionarios o empleados de su departamento las funciones que por la presente ley se le transfieren; para suprimir las Juntas y Tribunales Examinadores del Estado Libre Asociado, excepto la Junta Examinadora de Aspirantes al Ejercicio de la Abogacía y la Comisión de Reputación de Abogados; para conceder ciertas facultades adicionales comunes a los Secretarios aquí designados y al Jefe del Servicio de Bomberos de Puerto Rico, o a las personas que ellos designen en relación a las nuevas funciones que por esta ley se les encomiendan y en relación a las personas que soliciten licencias y a los profesionales y artesanos ya licenciados; para asignar fondos a cada uno de los departamentos gubernamentales para desempeñar las funciones adicionales que se le

asignan por esta ley; para derogar la ley creando la "Oficina Administrativa de Juntas Examinadoras y Juntas de Registro"; para fijar el 1ro. de enero de 1956, como fecha de vigencia de esta ley.

EXPOSICION DE MOTIVOS

Habiendo el pueblo de Puerto Rico determinado su organización sobre una base democrática para asegurar los derechos humanos a todas las personas por igual y siendo el espíritu del Estado Libre Asociado establecer los mismos derechos para todos a través de la Legislatura de Puerto Rico y legislar sus leyes sin que se establezcan privilegios ni favoritismos que puedan monopolizar el derecho al trabajo perjudicando el derecho de toda persona preparada y capacitada a ejercer libremente su profesión u oficio, es necesario suprimir todas las Juntas y Tribunales Examinadores del Estado Libre Asociado de Puerto Rico, con excepción de los organismos que regulan el ejercicio de la abogacía, función ésta puramente judicial, y establecer que todas las profesiones y oficios sean controlados y regulados por los Secretarios y Jefes de los Departamentos y agencias gubernamentales del Estado Libre Asociado, ejercitando dichos funcionarios todas las facultades y deberes que al amparo de las leyes orgánicas, según enmendadas, ejercen las Juntas y Tribunales Examinadores en Puerto Rico, así como los presidentes y miembros de dichos organismos, para asegurarle al pueblo de Puerto Rico mayor protección a la vida, la salud y la prosperidad y eliminar de una vez y para siempre los privilegios establecidos por grupos que en el pasado han sido muy funestos para la buena marcha del pueblo.

Basado en este principio le corresponde a la Legislatura de Puerto Rico, compuesta de hombres libres representando a su pueblo, corregir los errores del pasado y determinar las mejores pautas para que los derechos sean para todos por igual según lo establece la Constitución del Estado Libre Asociado de Puerto Rico, y por tanto:

Decrétase por la Asamblea Legislativa de Puerto Rico:

Artículo 1.—El título corto de esta ley será "Ley para Transferir las Funciones de las Juntas y Tribunales Examinadores de Puerto Rico."

Artículo 2.—Los términos "Secretario", "Secretario de Gobierno" y "departamento" y "departamentos gubernamentales" incluyen, a los fines de esta ley, al Servicio de Bomberos de Puerto Rico y a su Jefe.

Artículo 3.—Por la presente se suprimen todas y cada una de las Juntas y Tribunales Examinadores del Estado Libre Aso-

ciado de Puerto Rico, excepto la "Junta Examinadora de Aspirantes al Ejercicio de la Abogacía", organizada por la Ley Núm. 17 de 10 de junio de 1939 y la "Comisión de Reputación de Abogados", organizada por la Ley de marzo 11 de 1909, y todas las funciones, facultades, poderes, derechos y deberes de dichas Juntas y Tribunales Examinadores suprimidos y de sus presidentes y miembros, se transfieren y serán ejercitados por los Secretarios de cada uno de los departamentos gubernamentales que a continuación se indican:

(A) Al Secretario de Salud se transfiere las funciones de las:

1. Junta Dental Examinadora, creada por la Ley Núm. 75, de 8 de agosto de 1925, según enmendada, incluyendo sus poderes al amparo de la Ley Núm. 6, de 8 de abril de 1954. (Higienista Dental).

2. Junta Examinadora de Enfermeras, creada por la Ley Núm. 77 de 1930, según enmendada, incluyendo sus poderes al amparo de la Ley Núm. 427 de 1946, según enmendada, (Escuela para el Adiestramiento de Enfermeras Auxiliares).

3. Tribunal Examinador de Médicos, creado por la Ley Núm. 22 de 22 de abril de 1931, según enmendada.

4. Junta Examinadora de Químicos, creada por la Ley Núm. 31 de 26 de abril de 1932, según enmendada.

5. Junta Examinadora de Optómetras de Puerto Rico, creada por la Ley Núm. 78 de mayo 15 de 1930, según enmendada.

6. Junta de Registro de Tecnólogos y Microscopistas, creada por la Ley Núm. 154 de 13 de mayo de 1939, según enmendada.

7. Junta Examinadora de Auxiliares Técnicos de Cirugía, creada por la Ley Núm. 288 de 15 de mayo de 1945, según enmendada, incluyendo los poderes sobre la Escuela Estadual de Auxiliares Técnicos de Cirugía, creada por la misma ley.

8. Junta de Farmacia de Puerto Rico, creada por la Ley Núm. 282 de 1945, según enmendada.

9. Junta Examinadora de Especialistas en Belleza, creada por la Ley Núm. 431 de 15 de mayo de 1950, según enmendada.

10. Junta Examinadora de Quiroprácticos, creada por la Ley Núm. 493 de 15 de mayo de 1952.

(B) Al Secretario de Obras Públicas se transfiere las funciones de las:

1. Junta Examinadora de Ingenieros, Arquitectos y Agrimensores, creada por la Ley Núm. 399 de 10 de mayo de 1951.

2. Junta Examinadora de Maestros y Oficiales Plomeros, creada por la Ley Núm. 88 de 4 de mayo de 1939, según enmendada.

(C) Al Secretario de Hacienda se transfiere las funciones de la:

1. Junta de Contabilidad, creada por la Ley Núm. 293 de 15 de mayo de 1945, según enmendada.

(D) Al Jefe del Servicio de Bomberos de Puerto Rico se transfiere las funciones de la:

1. Junta Examinadora de Operadores de Máquinas Cinematográficas y Peritos Electricistas, creada por la Ley Núm. 13 de julio 3 de 1923, según enmendada.

(E) Al Secretario del Trabajo se transfiere las funciones de la:

1. Junta Examinadora de Trabajadores Sociales, creada por la Ley Núm. 171 de 11 de mayo de 1940, según enmendada.

(F) Al Secretario del Departamento de Agricultura y Comercio se transfiere las funciones de las:

1. Junta Examinadora de Veterinarios, creada por la Ley Núm. 59 de 13 de abril de 1916, según enmendada.
2. Junta Examinadora de Agrónomos, creada por la Ley Núm. 20 de 9 de abril de 1941, según enmendada.

(G) Al Secretario de Instrucción Pública se transfiere las funciones de la:

1. Junta Examinadora de Anunciadores Profesionales, creada por la Ley Núm. 123 de 13 de mayo de 1937.

Los Secretarios anteriormente mencionados desempeñarán todas las funciones de las Juntas y Tribunales Examinadores aquí especificados que estos organismos desempeñan al amparo de cualquier otra legislación, en adición a las leyes relacionadas en el primer párrafo de este artículo.

Artículo 4.—Los Secretarios de cada uno de los departamentos gubernamentales designados en el Artículo 3 estarán autorizados para delegar todas o algunas de las facultades, derechos y funciones que por esta ley se les confieren en cualquier funcionario o empleado de su departamento que cada uno designe, ex-

cepto que no podrá delegar la función de aprobar, enmendar y derogar reglamentos.

Artículo 5.—Todos los archivos, documentos, records, sellos y propiedades de todas y cada una de las Juntas y Tribunales Examinadores que por la presente se suprimen, en poder de los presidentes y miembros de cada uno de dichos organismos y en poder del Secretario de Estado, como sucesor de las funciones de la antigua "Oficina Administrativa de Juntas Examinadoras y Juntas de Registro", a virtud del Artículo I del Plan de Reorganización Núm. 7 de 1950, se entregarán al Secretario del departamento gubernamental a quien se transfieren las funciones de cada una de las Juntas y Tribunales Examinadores, según se dispone en el Artículo 3 de esta ley.

Artículo 6.—Cuando los Secretarios procedan a nombrar el personal necesario para desempeñar las funciones que por esta ley se les encomienda, dichos funcionarios darán preferencia en tales nombramientos a los empleados y funcionarios que actualmente desempeñan puestos en el Departamento de Estado, a cargo de las funciones de la antigua "Oficina Administrativa de Juntas Examinadoras y Juntas de Registro", trasladándose a dichos empleados y funcionarios a los distintos departamentos gubernamentales con los mismos sueldos, calificaciones, categorías y funciones que actualmente desempeñan.

Artículo 7.—Por la presente se declara que todas las leyes reglamentando las distintas profesiones y oficios quedan en vigor como reglamentos básicos de los Secretarios correspondientes, en adición a los ya existentes adoptados y promulgados por cada una de las Juntas y Tribunales Examinadores, y como tales podrán ser enmendados de acuerdo con el Artículo 10, inciso D.

Artículo 8.—Todas las reglas y reglamentos aprobados por todas las Juntas y Tribunales Examinadores en Puerto Rico continuarán en toda su fuerza y vigor hasta tanto los correspondientes Secretarios de los departamentos gubernamentales a que se hacen referencia en esta ley, modifiquen, enmienden o deroguen total o parcialmente dichos reglamentos, modificaciones, enmiendas o derogaciones que deberán ser refrendadas según se dispone en el párrafo "D" del Artículo 10 de esta ley.

DISPOSICIONES GENERALES EN RELACION A LOS SECRETARIOS, A LAS PERSONAS QUE SOLICITAN LICENCIAS O PERMISOS, Y A LOS PROFESIONALES Y ARTESANOS YA LICENCIADOS.

Artículo 9.—En adición a todos los derechos, poderes, facultades y deberes que por la ley regulando cada una de las Juntas y Tribunales Examinadores, según enmendadas, se conceden a

éstas y a los presidentes y a los miembros de dichas Juntas y Tribunales, (leyes que quedan en vigor como reglamentos básicos de los distintos Secretarios), funciones que por la presente ley se transfieren a los Secretarios de gobierno designados en el Artículo 3 de esta ley, dichos Secretarios y los funcionarios designados por éstos, tendrán los siguientes poderes y facultades:

A Podrán requerir, para la preparación, celebración y corrección de los exámenes y la preparación de los reglamentos a ser adoptados por los Secretarios, la cooperación de los miembros de las facultades de la Universidad de Puerto Rico, así como la cooperación de los profesionales, obreros y artesanos previamente licenciados y que hayan ejercido la profesión u oficio por un período no menor de cinco (5) años y que trabajen para el Estado Libre Asociado de Puerto Rico;

B Aprobar todas las reglas y reglamentos que sean necesarios y convenientes para el desempeño de las funciones que por la presente ley se les transfieren y para la concesión de licencia y toda otra clase de permiso a los candidatos. Esta facultad incluye la de enmendar, derogar y sustituir en cualquier forma, total o parcialmente, los reglamentos básicos de los Secretarios y los vigentes reglamentos aprobados por cada una de las diferentes Juntas y Tribunales Examinadores que por la presente ley se suprimen.

(1) Una vez que un Secretario enmiende, sustituya o derogue cualquier parte de un reglamento básico, o de un reglamento vigente a la aprobación de esta ley, aprobado por una Junta o Tribunal Examinador, o adopte un nuevo reglamento en sustitución de dichos reglamentos o enmiende estos últimos reglamentos, dicho funcionario remitirá suficientes copias a la Asamblea Legislativa para su aprobación en cualquier sesión ordinaria, y la Asamblea Legislativa podrá enmendarlos, modificarlos o tomar cualquier otra determinación mediante Resolución Concurrente.

(2) Si la Asamblea Legislativa no tomare acción alguna durante la sesión ordinaria en que fueren presentados, dichos reglamentos o las enmiendas a los mismos entrarán en vigor inmediatamente que dicho cuerpo legislativo recese, y los mismos tendrán fuerza de ley.

(3) Una vez los reglamentos o las enmiendas a los mismos entren en vigor, serán publicados en dos periódicos de circulación general tres veces en un período de treinta (30) días.

Artículo 10.—En adición a las causales especificadas por cada uno de los reglamentos básicos de los Secretarios, cualquier licencia ya expedida para ejercer cualquier profesión u oficio en

Puerto Rico y las que en el futuro se expidan, podrá ser suspendida o cancelada por un Secretario, si éste determinara que ha habido prueba suficiente de que el tenedor de dicha licencia ha incurrido en cualquiera de las infracciones siguientes:

(a) Que ha sido convicto de un delito grave;
(b) Que se dedica al uso habitual de drogas o bebidas intoxicantes;
(c) Que ha obtenido su licencia por medio de engaño, fraude o falsa representación;
(d) Que ha anunciado o publicado voluntariamente manifestaciones falsas, fraudulentas o conducentes a engaño en lo que respecta a su arte, habilidad o conocimiento o a sus métodos y sistemas de trabajo en otras profesiones;
(e) Que ha ingresado en cualquier entidad, sociedad o agrupación que sea subversiva para derrocar el Gobierno de los Estados Unidos de América o al Gobierno del Estado Libre Asociado de Puerto Rico a la fuerza.

Artículo 11.—Cualquier persona podrá presentar ante cualquier Secretario de un departamento gubernamental, cargos bajo juramento contra cualquier profesional o artesano por violación a las disposiciones de los reglamentos básicos de los Secretarios y cualquier otro reglamento, o por haber cometido cualquier delito o realizado cualquier infracción a lo estipulado en el Artículo 10 de esta ley, así como por cualquier actuación ilegal o inmoral. Sometidos los cargos el Secretario celebrará la correspondiente audiencia, luego de citar a la parte perjudicada con no menos de diez (10) días de anticipación. El Secretario deberá resolver si los cargos han sido probados, y en caso afirmativo, procederá a cancelar la licencia a dicho profesional o artesano. La decisión del Secretario será revisable por el Tribunal Superior de Puerto Rico mediante "certiorari", en el cual sólo serán revisables cuestiones de derecho, y el cual deberá ser interpuesto dentro de treinta (30) días a partir de la notificación a la parte perdidosa del fallo del Secretario.

Artículo 12.—El procedimiento estatuído en los Artículos 10 y 11 regirá la suspensión y cancelación de licencias ya expedidas y que se expidan en el futuro, a partir de la vigencia de esta ley, irrespectivamente de cualquier otra disposición en contrario que establezcan los reglamentos básicos de los Secretarios a que se hace referencia en el Artículo 7 de esta ley.

Artículo 13.—En adición a las distintas penalidades que se establecen en cada uno de los reglamentos básicos de los Secretarios, y en los otros reglamentos, será ilegal para cualquier persona el practicar u ofrecer practicar una profesión en Puerto Rico sin estar oficialmente autorizada por el correspondiente Secre-

tario, o por el delegado designado por dicho Secretario. La violación a este artículo será castigada como un delito menos grave.

Artículo 14.—Por la presente se asigna de cualesquiera fondos en el Tesoro Estatal la suma total de veinticinco mil (25,000) dólares que será puesta a la disposición de los departamentos gubernamentales que a continuación se indican y en las cantidades que a continuación se especifican:

A—Al Departamento de Salud, $15,000;

B—Al Departamento de Obras Públicas, $2,500;

C—Al Departamento de Hacienda, $1,250;

D—Al Servicio de Bomberos de Puerto Rico, $1,250;

E—Al Departamento del Trabajo, $1,250;

F—Al Departamento de Agricultura y Comercio, $2,500;

G—Al Departamento de Instrucción Pública, $1,250.

Artículo 15.—Por la presente se deroga la ley que organiza la Oficina Administrativa de Juntas Examinadoras y Juntas de Registro, Ley Núm. 320, de 13 de abril de 1946, disponiéndose que esta ley quedará derogada el día primero de enero de 1956.

Artículo 16.—Si cualquier cláusula, párrafo o artículo o parte de esta ley fuera declarado inconstitucional por un Tribunal de jurisdicción competente la sentencia a tal efecto no invalidará el resto de esta ley.

Artículo 17.—Esta ley empezará a regir el día primero de enero de 1956.

Artículo 18.—No obstante el hecho de que esta ley entrará en vigor el día primero de enero de 1956, por la presente se autoriza a los distintos Secretarios de los distintos departamentos gubernamentales y al Secretario de Estado, Sucesor de las funciones de la Oficina Administrativa de Juntas Examinadoras y Juntas de Registro, para que, a partir de la aprobación de esta ley por el Gobernador de Puerto Rico, y a virtud de lo estatuído en este artículo, procedan a hacer los arreglos previos necesarios de manera de que no haya interrupción alguna en la expedición de licencias a profesionales y artesanos,

SENADO DEL ESTADO LIBRE ASOCIADO DE PUERTO RICO

Comisión para el Estudio y Revisión de las Leyes

Organizando o Colegiando Profesiones, Oficios y Negocios.

San Juan, Puerto Rico,
26 de abril de 1955.

AL SENADO:

Vuesta Comisión, después de haber estudiado el P. del S. 511 y haber considerado **un informe sobre las Juntas y Tribunales Examinadores y los distintos Colegios Profesionales preparado por la Oficina de Servicios Legislativos,** ha decidido recomendar la aprobación de un Sustituto a dicho proyecto en que se incorporan las enmiendas que la Comisión considera necesarias para resolver fundamentalmente los males de que adolecen las actuales Juntas y Tribunales Examinadores. El Sustituto que se recomienda solamente afecta a las Juntas y Tribunales Examinadores de Puerto Rico, y en nada interviene con los Colegios Profesionales existentes en el Estado Libre Asociado de Puerto Rico.

El Sustituto recomendado suprime todas y cada una de las Juntas y Tribunales Examinadores del Estado Libre Asociado, con una excepción, la cual mencionamos más adelante, y transfiere todas sus funciones a los distintos Secretarios de Gobierno y al Jefe del Servicio de Bomberos de Puerto Rico, según se indica en el Artículo 3 del Sustituto, funciones que también podrán ser desempeñadas por el funcionario o empleado que el correspondiente Secretario designe, con la única limitación de que dicho funcionario o empleado designado no podrá ejercer la facultad de aprobar reglamentos. Se deroga la ley que organiza la Oficina Administrativa de Juntas Examinadoras y Juntas de Registro, (Ley Núm. 320 de 1946), oficina que fué suprimida por el Artículo I del Plan de Reorganización Núm. 7 de 1950, y sus funciones transferidas al Secretario Ejecutivo de Puerto Rico, hoy Secretario de Estado, ya que las funciones de este funcionario se transfieren a los diferentes Secretarios.

El Sustituto no afecta el funcionamiento de la Junta Examinadora de Aspirantes al Ejercicio de la Abogacía, organizada por la Ley Núm. 17 de 10 de junio de 1939, ni el de la Comisión de Reputación de Abogados, creada por la Ley de 11 de marzo de 1909. Estos dos organismos quedan exentos de las disposiciones del Sustituto por ser éstos los que regulan la profesión de la abogacía, función netamente judicial que ejerce el Tribunal Supremo de Puerto Rico, y la cual no puede ser controlada por el Poder Legislativo.

A virtud de las disposiciones del Sustituto, las leyes vigentes que regulan las distintas Juntas y Tribunales Examinadores que-

dan en vigor como reglamentos básicos de los Secretarios, y éstos ejercerán las funciones que se les transfieren de acuerdo con las mismas. Asimismo, quedan en vigor todas las reglas y reglamentos adoptados por las distintas Juntas y Tribunales Examinadores, vigentes en la actualidad, hasta tanto los Secretarios enmienden, modifiquen o deroguen los mismos, todo ello mediante el procedimiento estatuído en el Artículo 9, letra B.

La idea que se ha tenido en mente es que dichas leyes, como reglamentos básicos de los distintos Secretarios y los otros reglamentos vigentes, continúen en vigor como hasta el presente, y que los Secretarios recomienden a la Asamblea Legislativa hacer en los reglamentos aquellos cambios que la experiencia demuestre que son necesarios, a los fines de mejorar la concesión de permisos y licencias a los profesionales y artesanos en Puerto Rico.

Entre las facultades adicionales comunes que se les confieren a los Secretarios por el Sustituto, quizás la más importante es la facultad de modificar, enmendar y derogar los reglamentos vigentes, incluyendo los reglamentos básicos del Secretario, así como aprobar, y enmendar nuevos reglamentos. Los reglamentos adoptados o enmendados por los Secretarios no podrán entrar en vigor hasta tanto sean remitidos a la Asamblea Legislativa, durante una sesión ordinaria, la cual tendrá facultad para modificarlos, enmendarlos, y tomar cualquier otra determinación sobre los mismos, mediante Resolución Concurrente. Si la Asamblea Legislativa no tomare acción sobre los mismos, los reglamentos y las enmiendas a éstos, entrarán en vigor al recesar la Asamblea, y éstos tendrán fuerza de ley.

El Sustituto también señala ciertas causales, en adición a las especificadas en las leyes que regulan las distintas Juntas y Tribunales Examinadores, (que a partir de la aprobación del Sustituto serán los reglamentos básicos de los distintos Secretarios), y las señaladas en los otros reglamentos, mediante las cuales los Secretarios podrán cancelar o suspender licencias ya concedidas o las que en el futuro se expidan. Disponen los Artículos 10, 11 y 12 que si un profesional o artesano violare alguna de dichas causales, cualquier persona podrá presentar cargos jurados contra el profesional o artesano ante el correspondiente Secretario, quien celebrará la correspondiente audiencia y decidirá sobre si los cargos han sido probados. En caso de que decida que se han probado los cargos, la persona perjudicada tendrá treinta (30) días dentro de los cuales podrá recurrir ante el Tribunal Superior, el que revisará la actuación del Secretario mediante certiorari en que sólo se podrá revisar cuestiones de derecho. Se dispone que este procedimiento de cancelar licencias expedidas será exclusivo y regirá desde la aprobación de la ley, irrespectivamente de cualquier otra

disposición en contrario que establezcan los reglamentos básicos de los Secretarios.

Para el desempeño de las funciones que por el Sustituto se transfieren a los Secretarios de los departamentos gubernamentales relacionados en el Artículo 3, a partir del 1º de enero de 1956, fecha en que comenzará a regir el Sustituto, si fuere aprobado, se asigna la suma de $25,000 distribuídos en la forma que se indica en el Artículo 14 del Sustituto.

La Comisión entiende que su trabajo no ha terminado y continuará ejerciendo sus labores a fin de poder dar mayor estudio a la forma en que dichos Colegios Profesionales deben ser reorganizados con el objeto de que dichas organizaciones no le deban su existencia a leyes de esta Asamblea Legislativa y sí a las actividades de los propios profesionales. A estos fines, la Comisión continuará sus actividades de manera que la misma pueda desempeñar totalmente la encomienda que se le asignara a través de la Resolución del Senado Núm. 36.

Respetuosamente sometido

Carlos Román Benítez,
Secretario.

Heraclio H. Rivera Colón,
Presidente



UNA MANERA SENCILLA DE ESTIMULAR LA



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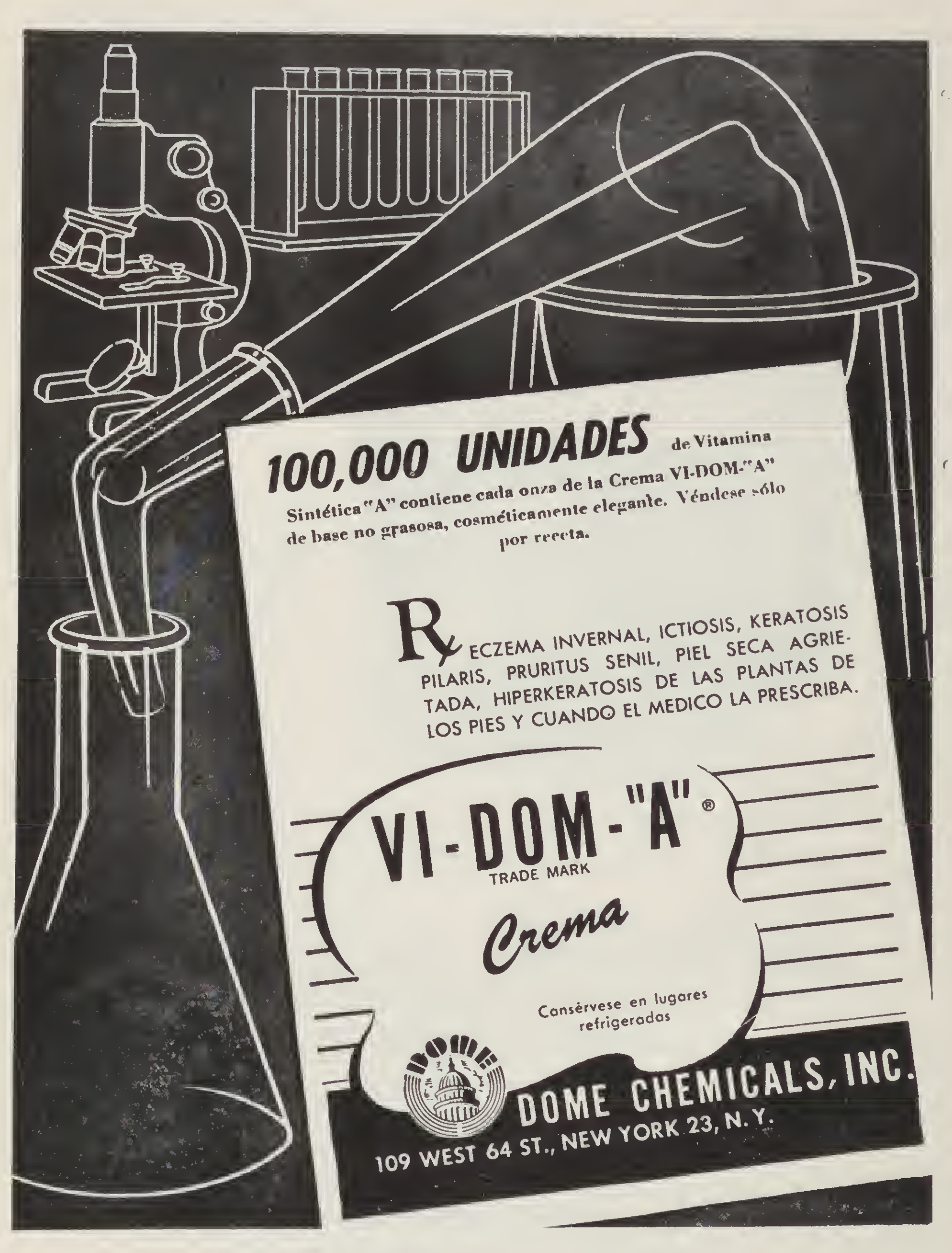
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BOLETIN

DE LA ASOCIACION MEDICA DE PUERTO RICO

VOL. 47 JULIO, 1955 No. 7

NEW RADIOPAQUE DIAGNOSTIC AGENTS

*MAURICE L. TAINTER, M.D.**

In recent years the continuous search in our laboratories for improved diagnostic agents has concentrated on the visualization of the gall bladder and ducts, the kidneys, lungs, cardiac chambers, the larger vessels, spinal canal and the vascular beds in the brain. It seems probable that the time is not far distant when all the hollow chambers of the body can be effectively visualized by X-ray with safety and with a high degree of diagnostic acumen. Space here does not permit discussion of each of these fields so that I will concentrate on the status of gall bladder diagnostic agents and of those which visualize the urinary tract through excretion urography.

Cholecystography: The recent major advance in the science of cholecystography was brought about through the intensive studies of Drs. Hoppe and Archer[1] in our Institute. These scientists, working for about five years, developed new and improved methods of synthesizing radiopaque materials and of studying them experimentally in the pharmacology laboratory. Their test procedures had such precision as to give, for the first time, reliable quantitative measures of the value of the various compounds studied. Archer synthesized nearly 150 radiopaque compounds which were potential gall bladder contrast media. Hoppe, in turn, studied these in experimental animals, determined their pattern of location in the gall bladder, their tolerance and safety.

Hoppe's method used cats as the primary animal for the study of these compounds. This animal was selected because a careful comparison by him of the response to radiopaque gall bladder dyes in various species revealed that the cat predicted more accurately the human response than did other animals, particularly the dog. It was found that some compounds which appeared to be very good in the dog were relatively inferior in humans. Too great reliance on dog data, therefore, has led some observers to draw unjustified conclusions as to the merits of the material in which they were interested.[2]

* Sterling-Winthrop Research Institute, Rensselaer, N.Y.

Hoppe's technic was to withdraw food from the cats for 24 hours, then to give them milk rich in cream, which emptied the gall bladder quickly. Six hours later the drug was administered by mouth. Pictures can be taken at various intervals, although he found that the most intense shadows were customarily seen at 18 hours. For the sake of quantitative comparison Hoppe graded the intensity of the shadows from 0 to 4, 4 being the most intense completely opaque gall bladder, as is illustrated in Figure 1.

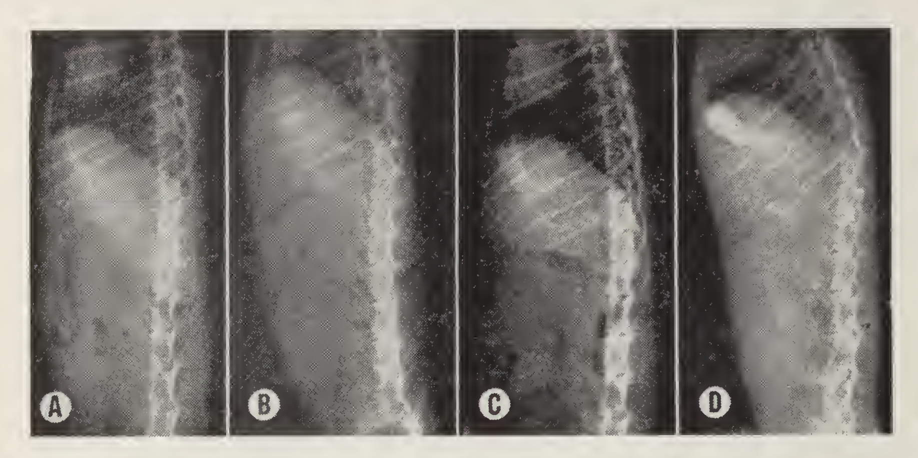


Fig. 1. Oral Telepaque(R) cholecystograms in the cat illustrating: (A) a poor (1), (B) fair (2), (C) good (3), and (D) an excellent (4) gall bladder visualization.

As a result of his extensive studies he determined that the most active compounds are those which are triiodoaminophenyl derivatives. These have a great advantage over molecules previously available, in that they contain three, rather than two, atoms of iodine, so that the best one of these contains 66.7% iodine. This compound, 3(3-amino-2,4,6-triiodophenyl)-2-ethylpropanoic acid,

I

$-CH_2CHCOOH$

CH_2

I - -I CH_3

NH_2

has been named Telepaque and given the common designation, iodopanoic acid.

Hoppe's and Archer's results have been summarized by them[1] in the data given in Table 1. It can be seen from these data that the compound, Win 2011, which later was named Telepaque, produced with doses of 100 mg./kg. a density of 3.6 units in Hoppe's

TABLE 1

CHOLECYSTOGRAPHIC AND TOXICOLOGIC PROPERTIES OF A SERIES OF 2-SUBSTITUTED-(3-AMINO-2,4,6-TRIIODOPHENYL)-PROPANOIC ACID DERIVATIVES IN EXPERIMENTAL ANIMALS

Compound	R (3-amino-2,4,6-triiodophenyl–CH_2–CH(R)–COOH)	Per Cent Iodine	Average Cholecystographic Index (ACI) in Cats at Oral Doses of (mg./kg.)*				Acute Oral Toxicity in mice $LD_{50} \pm$ s.e. gm./kg.	Relative Activity at 100 mg./kg.: Iodoalphionic acid = 100
			25	50	100	200		
Iodoalphionic acid	–	51.5	1.4	2.1	2.5	3.3	3.8 ± 0.3	100
WIN 4837	H	70.1	0.6	0.8	1.4	2.2	>3.2 at 24 hr. 1.4 at 7 days	56
WIN 4134	$-CH_3$	68.4	1.0	1.9	2.9	4.0	8.0 at 24 hr. 6.0 at 7 days	115
WIN 2011 (Telepaque(R))	$-CH_2CH_3$	66.7	1.6	2.6	3.6	—	15.8 ± 1.1	144
WIN 4414	$-CH_2CH_2CH_3$	65.1	0	0.4	1.2	1.8	>6.4	48
WIN 4152	$-CH_2CH_2CH_2CH_3$	63.6	0.2	0.4	0.4	1.8	—	16

* Average of 5 cats per dose level except with iodoalphionic acid and Telepaque(R) where 20 cats were used per dose level.

ACI = Average Cholecystographic Index, representing the average numerical score for 5 or more observations at a given dose level.

scale, whereas iodoalphionic acid (Priodax), the compound most commonly used previously, produced a density of only 3.3 units when given in twice the dose. Hoppe's measurements of the density showed that Telepaque produced 44% more intense shadows than Priodax regardless of dosage. One might expect that the increased iodine content and concentration in the bile of Telepaque would be associated with increased toxicity. However, the data revealed quite the contrary; for, while Priodax was fatal in doses of 3.8 gm./kg., 15.8 gm. of Telepaque were required for the same degree of effect. In other words, Telepaque, although 44% more effective as a radiopaque, was only one-fourth as toxic.

These animal tests have proved to be almost completely transferable to patients. Thus, Morgan & Stewart[4] in a quantitative study of the density of the shadows in patients found that when patients were given both compounds, Telepaque produced shadows 35% more dense than those of Priodax, a figure which coincides remarkably well with that observed in the animal laboratory. Morgan & Stewart also observed in those cases where visualization failed or was incomplete and readministration of the drug was required to make the diagnosis more certain, Telepaque had to be readministered in 40% fewer patients than did Priodax in order to obtain reliable results.

The kind of pictures observed in patients with Telepaque are illustrated by some of the figures from typical patients. It will be noted that frequently the density is such that the biliary ducts are adequately visualized throughout their length. The common duct and the cystic duct are seen very frequently, and often even some branches of the intrahepatic ducts can be discerned as well. Figure 2 shows a gall bladder containing one opaque stone. In Figure 3 are shown three radiolucent stones. In Figure 4 is depicted a patient after 6 tablets of Telepaque who had a gall bladder containing many translucent stones. This picture was taken prone. When this patient was stood upright the stones floated into a horizontal layer, as is shown in Figure 5. After a fat meal the cystic and common ducts were visualized, although the gall bladder did not empty vigorously. (Figure 6). Another film which illustrates the good visualization of the ductal system frequently obtained is given in Figure 7. Here, 12 minutes after a fat meal the gall bladder is actively contracting, and the cystic, common and hepatic ducts can be readily distinguished,

In a series of 1000 patients Whitehouse and Martin[3] have given a very informative analysis of the quality of visualization produced by Telepaque as contrasted with Priodax.

Excellent visualization was produced in 59.6% of the Telepaque and 20.2% of the Priodax patients. At the opposite end of

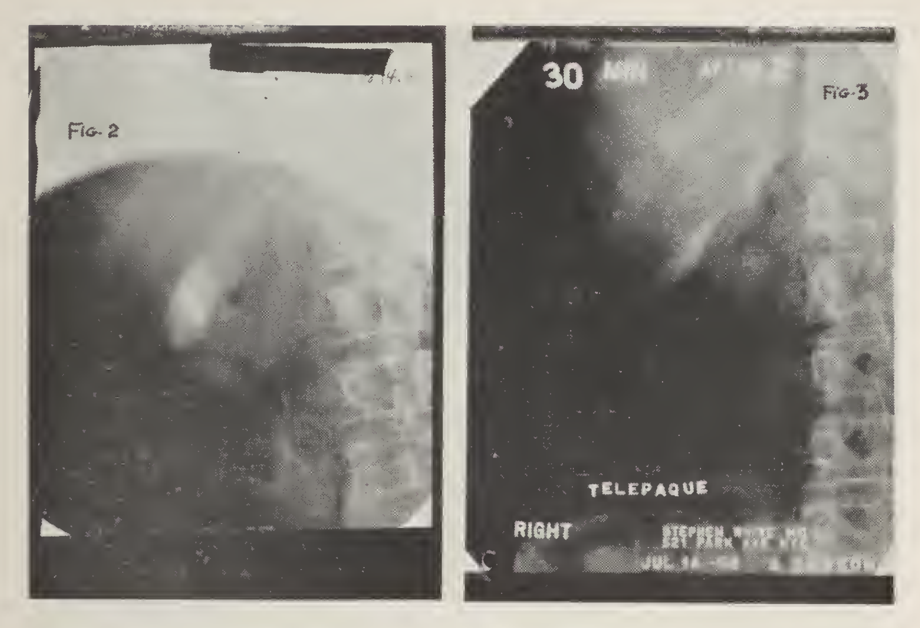


Figure 2. Cholecystogram with 6 tablets of Telepaque in 71 year old woman, showing well calcified gall bladder with single calcified stone.

Figure 3. Cholecystogram with 6 tablets of Telepaque showing three translucent stones in the gall bladder (Courtesy of Dr. Stephen White, New York).

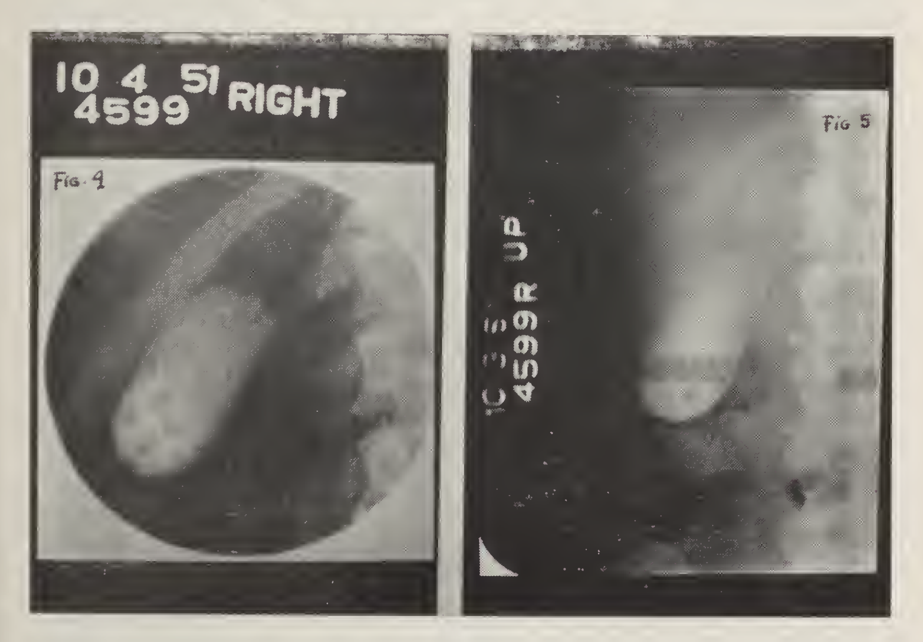


Figure 4. Cholecystogram after 6 tablets of Telepaque, showing multiple translucent stones. Patient in prone position.

Figure 5. Same patient as Figure 4, in the upright position. Stones have collected in a horizontal layer.

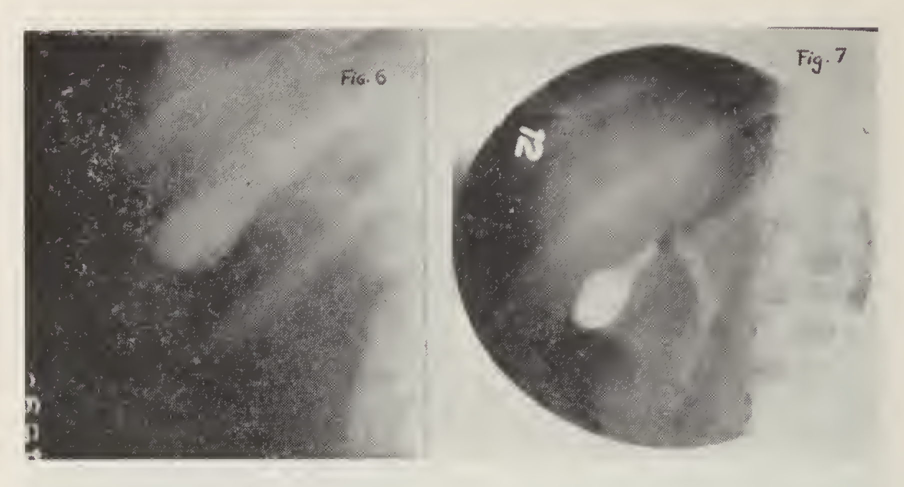


Figure 6. Same patient of Figures 4 and 5, 40 minutes after a fat meal. The gall bladder is only partially emptied, but the cystic and common ducts are visualized.

Figure 7. Telepaque 6 tablets. Picture made 12 minutes after a fat meal. Shows contraction of gall bladder and visualization of the ductal system. (Courtesy of Dr. Louis P. Divilio).

the scale, no visualization or faint pictures were seen in 13.2% and 21.8% respectively, of the patients given Priodax as compared to 9.4% and 7.6% with Telepaque. These authors conclude that "the use of Telepaque resulted in nearly three times as many excellent shadows as did Priodax, and the number of cases with faint or no shadows was demonstrably less."

Side Effects: With a diagnostic procedure such as visualizing the gall bladder, an important consideration always must be whether there are side effects experienced by the patient which make the procedure less than completely satisfactory. The animals studied were remarkably free from any such effect which might be of clinical significance. However, they are not necessarily conclusive witnesses to whether there would be effects in patients.

We have tried to assemble the data which have been published on the three leading oral gall bladder compounds. These are the Telepaque and Priodax already referred to, and a newer compound, Teridax, also known as iophenoxic acid, which has become available so recently that only a few data are available. However, we have been able to assemble from the published literature, in a form which allows us to summarize the clinical responses to them, 1801 patients on Telepaque, 4492 on Priodax, and 150 on Teridax. In Table 3 the number of cases are reported as the denominator in which the presence or absence of a symptom was specifically indicated and the number of positive reactions is indicated as the numerator of the fraction. Obviously, where a

TABLE 2

QUALITY OF VISUALIZATION WITH PRIODAX AND TELEPAQUE
(Data of Whitehouse and Martin (3)

	Priodax 500 Cases	Telepaque 500 Cases
Excellent Visualization		
1. No stone	98 (19.6%)	270 (54.0%)
2. Radiolucent stone	2 (0.4%)	25 (5.0%)
3. Opaque or partially opaque stone	1 (0.2%)	2 (0.4%)
4. ? Polyp	0	1 (0.2%)
TOTAL	101 (20.2%)	298 (59.6%)
Good Visualization		
1. No stone	191 (38.2%)	97 (19.4%)
2. Radiolucent stone	16 (3.2%)	19 (3.8%)
3. Opaque or partially opaque stone	2 (0.4%)	1 (0.2%)
TOTAL	209 (41.8%)	117 (23.4%)
Faint Visualization		
1. No stone	85 (17.0%)	24 (4.8%)
2. Radiolucent stone	16 (3.2%)	9 (1.8%)
3. Opaque or partially opaque stone	8 (1.6%)	5 (1.0%)
TOTAL	109 (21.8%)	38 (7.6%)
Non-visualization		
1. No stone	75 (15.0%)	44 (8.8%)
2. Opaque or partially opaque stone	6 (1.2%)	3 (0.6%)
TOTAL	81 (16.2%)	47 (9.4%)

symptom was not specifically mentioned by the observers it is likely that it was not present or was insignificant. Nevertheless, these have not been considered negative cases and added in unless there was specific mention of the observation in question. Hence, the reliability of some of the percentages quoted has to be weighed in the light of the number of cases represented by the observations.

TABLE 3

Comparison of Side Effects Reported in 1801 Patients Receiving Telepaque, 4492 Patients Receiving Iodoalphionic Acid & 150 Patients Receiving Iophenox Acid (References 3 to 27) in Full Doses.

	Telepaque (Iopanoic Acid)		Priodax (Iodoalphionic Acid)		Teridax (Iophenoxic Acid)	
	No.	%	No.	%	No.	%
Total Cases	1801		4492		150	
Patient with undesirable symptoms	279/951	29.3%	747/1675	44.6%	18/50	36 %
Dry mouth or throat	1/83	1.2%	3/100	3 %		
Heartburn	2/95	2.1%	2/112	1.8%		
Burning on swallowing	15/310	4.8%	24/120	20 %		
Pain	0/50	0%	108/1722	6.3%	3/50	6 %
Nausea	171/1636	10.5%	781/4342	18 %	31/150	20.5%
Vomiting	10/1636	0.6%	101/4179	2.4%	4/100	4 %
Cramps	30/195	15.4%	23/212	10.8%	25/100	25 %
Diarrhea	330/1540	21.4%	1090/4242	25.7%	41/150	27.3%
Headache	39/395	9.9%	33/412	8.0%	20/100	20 %
Faintness and Dizziness	19/295	6.4%	18/312	5.8%	24/100	24 %
Pruritus	0/310	0 %	2/120	1.7%		
Skin eruption	0/310	0 %	3/120	2.5%		
Good Emptying with Fat Meal	105/114	92.1%	64/100	64 %		
Residual Opacity in GI Tract	163/183	89.1%	14/100	14 %	11/50	22 %
Dysuria	126/1396	9.0%	592/4124	14.4%	36/100	36 %

In general, it can be seen that 29.3% of the patients had some kind of undesirable symptom when Telepaque was given, and that this figure was 44.6% with Priodax, and 36% with Teridax. Thus it appears that adverse symptoms are considerably less common with Telepaque than with the other agents. The nature of these adverse effects varied from patient to patient. Nausea was present in only 10.5% of the Telepaque patients as compared to 18% of those having Priodax, and 20.5% of those receiving Teridax. Vomiting was a very infrequent result from Telepaque, being seen in only 0.6%. With Priodax, vomiting was 4 times as frequent, being 2.4%, and with Teridax was about 7 times as frequent, being 4%. Diarrhea is caused by these radiopaques probably more frequently than any other symptom. It was present in 21.4% of the Telepaque, 25.7% of the Priodax, and 27.3% of the Teridax patients. Where direct comparisons of the compounds were made, the comment was drawn, however, that the diarrhea from Telepaque was less severe than that from Priodax, quite apart from the absolute incidence.

Headaches from these drugs are probably coincidental rather than being produced by any causal mechanism. This symptom was observed in 9.9% and 8% respectively, of the Telepaque and Priodax patients. However, it was present in 20% of the Teridax patients, which raises a suspicion that this latter compound may have some specific influence in producing headaches not possessed by the other two preparations. Distress in the gastro-intestinal tract associated with swallowing the drug is reported by various observers under the headings of heartburn, pain, burning on swallowing and cramps. The pattern of these varied with the compound and, to a large extent, with the observer. Priodax seemed to be the most free of producing gastro-intestinal cramps while Telepaque and Teridax in the order named were not so good in this respect. However, Priodax caused 20% incidence of burning on swallowing, a symptom which was only reported in 4.8% of the Telepaque patients. When these results are lumped together to get a rough estimate of the overall gastric tolerance of the compounds, it is found that Telepaque produced these symptoms in a significantly smaller proportion than either Priodax or Teridax.

Disorders of the skin were not observed in any patients receiving Telepaque; however, 2.5% of those getting Priodax had skin eruptions and 1.7% had pruritus. This might indicate that Telepaque has a lessened chance of producing allergic reactions than the older preparation.

There was a marked difference between these products in the amount of residual radiopaque particles visualized in the gastrointestinal tract. Every radiologist has observed that when large

amounts of radiopaque material are secreted into the duodenum in the bile there may be a diffuse cloud cast over the viscus. However, with Telepaque there is more than this excretory opacity in the gut, since there were visualized in 89% of the patients small particles of apparently unabsorbed tablets. These are usually sufficiently far from the gall bladder shadow to cause no confusion in the interpretation of calculi in the biliary tract. In case of suspicion, taking the picture from the proper angle will quickly determine whether or not such particles are within the gall bladder.

Where a gall bladder fails to visualize, it may be very comforting to the radiologist and surgeon to see these particles in the gastro-intestinal tract, because then they can be sure that the patient has swallowed the tablets as instructed and that the failure to visualize is due to actual functional defect and not to lack of patient cooperation. Cholecystectomies have been inadvertently scheduled on supposedly non-functioning gall bladders, only to discover later that the organ did not visualize because the patient neglected to swallow the drug as instructed, or perhaps vomited it and, therefore, did not absorb the material. What might, thus, be considered as a defect in the performance of the Telepaque, in reality becomes an asset of considerable value in its clinical use.

Another aspect of the action of these compounds is the information that may be drawn as to whether the gall bladder is able to empty actively under the normal stimulus or not. A gall bladder which does not empty freely when a proper fat meal is given may well be suspected of being physiologically abnormal and, therefore, may be more likely to require surgical intervention. Hence, there is interest in how frequently the gall bladders will empty under the stimulus of a fat meal. The table shows that out of 114 patients in whom this facet was commented upon, 105 had good emptying of the gall bladder after the fat meal when given Telepaque, or an emptying rate of 92.1%. With Priodax, however, the emptying rate in an entirely similar series of patients was only 64%. If it can be assumed that the poorer emptying after Priodax was apt to be interpreted as evidence of biliary dysfunction, then it is likely that diagnostic errors may have been made because of the failure of the Priodax-treated gall bladder to empty properly under the stimulus of the fat meal.

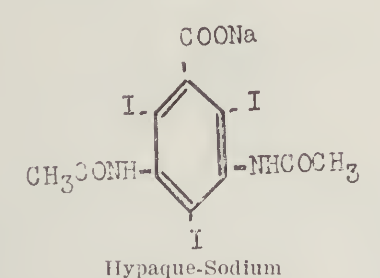
SUMMARY

These observations on Telepaque and related compounds may be summarized by saying that this new compound gives a higher percentage of excellent and good visualization of the gall bladder

than do the compounds heretofore available. It also produces a more dense picture which permits a higher percentage of critical diagnoses, particularly in those patients where the other agents give equivocal answers. As a result of these more informative results, re-administration of the gall bladder dye is decreased in frequency and reliable diagnoses are made on the first examination much more often. This improved diagnostic reliability of Telepaque is associated with a lower incidence of side effects and with less intensity of those side effects which are seen. The conclusion, therefore, seems appropriate that with this compound better gall bladder pictures can be obtained than previously and that the entire science of cholecystographic diagnosis has been made more accurate.

EXCRETION UROGRAPHY

The same kind of attack on the problems of excretion urography as has just been described for cholecystography has been carried out by Drs. Larsen and Hoppe[32] in our Institute. Dr. Larsen in a brilliant series of syntheses finally evolved a compound which was excreted quantitatively by the kidneys and which contained 3 atoms of iodine just as does Telepaque. This gave the new compound an iodine content of almost 60%. The substance is a benzoic acid derivative, having the chemical formula sodium 3-5-diacetamido-2,4,6-triiodobenzoate.



Hypaque-Sodium

It has the important physical property of being neutral in concentrated solutions, so that large amounts of it can be given intravenously without disturbing the acid-base balance of the blood. This compound, known as Hypaque Sodium, has been given the general name of diatrizoate sodium and is now available for excretion urography in the form of a 50% solution.

This material is excreted in the urine beginning almost immediately after its intravenous injection. The kidneys concentrate the solution to a very high degree and, hence, stand out in X-ray pictures with a brilliance not available previously. As a matter of fact, in some particularly favorable patients even the collecting tubules of the kidneys can be visualized, thus allowing better in-

sight into the position and appearance of the pyramids. Illustration of its excretion by the kidneys of rabbits, taken from Hoppe's laboratory studies is given in Figures 8, 9 and 10.

In spite of the very high iodine content of this compound it is one of the most inert drugs, in the pharmacodynamic sense, known. It causes so little disturbance of blood pressure and respiration that between 10 and 14 gm./kg. intravenously are required in laboratory animals for fatal effects. This gives it a very wide margin of safety as compared to the doses which are used in patients for diagnosis.

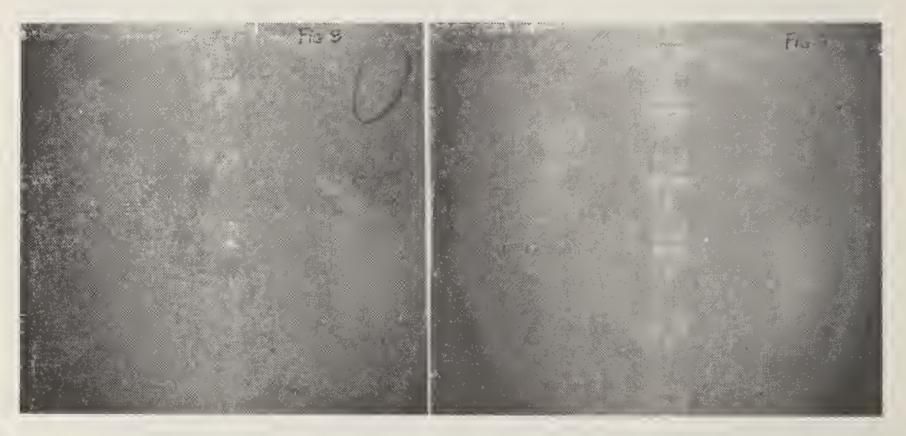


Figure 8. Control flat plate of rabbit before Hypaque injection (Courtesy of Dr. J. O. Hoppe).

Figure 9. Flat plate of rabbit abdomen 5 minutes after 400 mgm./kg. Hypaque Sodium intravenously (Courtesy of Dr. J. O. Hope).

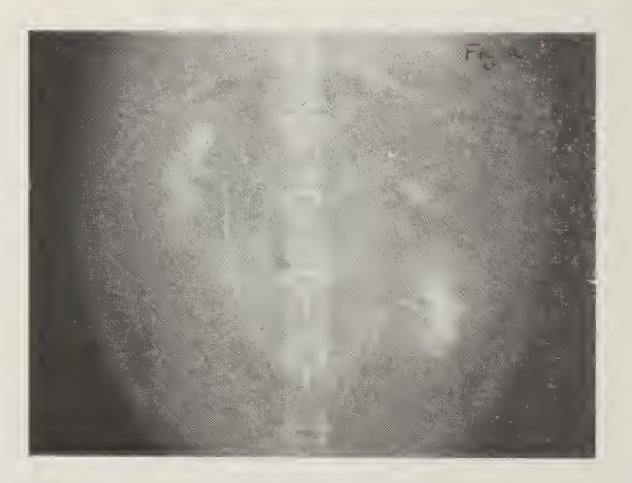


Figure 10. Same rabbit as in Figure 8 and 9, at 10 minutes after injection.

The remarkable visualization of the kidneys following intravenous injection of Hypaque is illustrated by plates from typical patients. Figure 11 is that of a 90 year old man who was being studied for hematuria. The film, made 25 minutes after injection of Hypaque, shows a normal upper urinary tract in which even the terminal collecting tubules of the renal papillae are visible. Hydronephrosis is shown in Figure 12. Note the dilated calyces and ureter and the heavy shadow (upper right segment) cast by the redundant ureter. In the next figure (No. 13) elongation and narrowing of the upper infundibulum are apparent as the result of a renal tumor (carcinoma) of the upper pole of the right kidney. A left renal tumor is depicted in Figure 14. Dr. Henry Bodner of Los Angeles, California, was able, with Hypaque, to prepare an excellent excretion urogram (Fig. 15) which clearly reveals the presence of double kidneys. In Figure 16 is illustrated the functional impairment of renal tuberculosis.

Although tens of thousands of injections of Hypaque Sodium have been made in the first months since the compound became available, only about 1,200 of these have been reported publicly so as to be available for analysis. In the first 1123 patients reported to us, as is summarized in Table 4, excellent and good diagnostic pictures were obtained in 85.3% of the cases, and fair

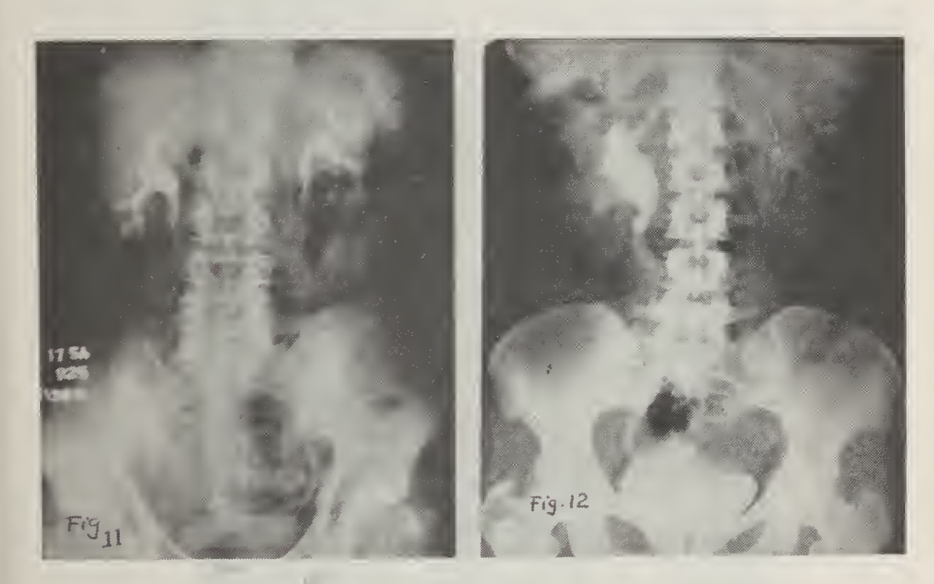


Figure 11. Male, 90 years - investigated for hematuria. Upper urinary tract normal. Terminal collecting tubes in renal papillae visualized. Film made 25 minutes after Hypaque injection.

Figure 12. Female, 49 years - right hydronephrosis, dilated calyces and ureter. Redundant ureter (upper right segment) casting heavy shadow.

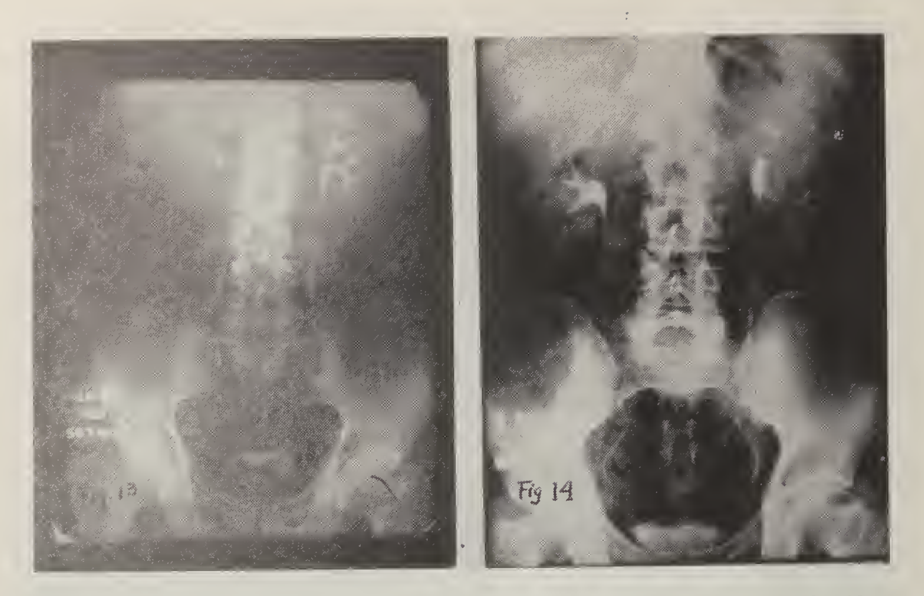


Figure 13. Male, 59 years - right renal tumor (carcinoma) upper pole. Note elongation and narrowing of upper infundibulum.

Figure 14. Male, 64 years - left renal tumor. Film made 25 minutes after Hypaque injection.



Figure 15. Male. Double kidneys. (Courtesy of Dr. Henry Bodner).



Figure 16. Woman, 17 years - evidence of impaired function on left 15 min. after i.v. Hypaque. Later films at 35 and 50 minutes indicate left renal tuberculosis with abscess at middle calyx.

pictures satisfactory for diagnosis in an additional 9.6%. Therefore, urograms suitable for clinical interpretation were secured in 94.9% of the administrations of Hypaque. In only 5.1% were pictures unacceptable for diagnosis. This is an extremely high level of clinical effectiveness which we believe surpasses that available heretofore.

Another question which arises is whether the use of the compound is associated with side effects which would make it less

TABLE 4

QUALITY OF UROGRAMS WITH HYPAQUE SODIUM
1123 PATIENTS (COOPERATIVE STUDY)

	No. Patients	Total
Satisfactory for diagnosis		
Excellent	569 (50.7%)	
Good	389 (34.6%)	
Fair	108 (9.6%)	
		1066 (94.9%)
Unsatisfactory for diagnosis		
Poor	57 (5.1%)	
		57 (5.1%)
		1123 (100%)

TABLE 5

CLINICAL RESPONSE IN 1203 PATIENTS RECEIVING HYPAQUE SODIUM FOR EXCRETION UROGRAPHY

	No. of Cases showing symptom out of 1203 patients	Percentage of cases showing symptom
No side effects	1087	90.3%
Nausea and vomiting	57	4.7%
Urticaria	7	0.58%
Vein cramps	12	1%
Mild vasomotor reaction	38	3.15%
Severe vasomotor reactions	2	0.16%

than completely satisfactory. A total of 1203 patients have been reported in whom conclusions regarding the side effects can be tabulated.[28-31] Of these, 90.3%, or 1087 out of the 1203, had no side effects at all. Vein cramps occurred in 1%.

Skin reactions, as demonstrated by urticaria, were present in 7 of the patients, or 0.58%. Presumably, this urticaria would be minimized in incidence or intensity by the administration of a suitable antihistaminic. Perhaps this is a point which will be investigated by some urologists who has a large enough series of patients to obtain accurate statistics on the factor. Gastro-intestinal symptoms were relatively uncommon, there being only nausea with occasional vomiting in 57 of the patients out of the 1203. This gives an incidence of gastro-intestinal distress of only 4.7%.

It is recognized that the most serious danger from excretion urographic compounds, in general, is vasomotor collapse. This has been experienced as marked flushing of the skin with fall of blood pressure coming after sensations of warmth or, in the more severe cases, as a fall of blood pressure to the shock level. In a few very rare cases with other compounds the reaction has ended fatally. Hypaque seems to be less afflicted with this kind of side effect than previously used compounds, although the incidence is sufficiently uncommon that enough clinical experience has not yet accumulated to permit a reliable estimate of the percentage incidence of such vasomotor effects. However, in the 1203 patients summarized here, 38 patients had mild vasomotor reactions of flushing and warmth which were transient and required no particular therapy. This was an incidence of 3.15% Two out of the entire series had temporary severe vasomotor reactions with blood pressure falling to dangerous levels. This calculates out as an incidence of 0.16%.

What this figure will be when 10,000 or more patients have been reported remains to be determined. Present indications are, however, that it will be a lower figure than that experienced previously with other agents. As a matter of fact, it seems that the low incidence of such reactions is quite remarkable. The dose of Hypaque is between 20 and 30 cc. of a 50% solution, and it is impressive evidence of its lack of toxicity that this tremendous weight of a foreign compound can be injected into the circulation within a very few minutes and still produce such a small incidence of untoward effects.

In summary, it might be concluded that in Hypaque, a new level of diagnostic reliability has been achieved for studying kidney function and pathology. The urologist has always been fortunate in being able to make the most accurate diagnoses of any of the medical specialties. He works with a precision of diagnosis and insight into the status of his patient, which unfortunately is not available to other branches of the healing arts. Therefore, its seems to us a contribution of considerable value to have been able to develop for the urologist a diagnostic agent, which, in this significant way, heightens still further his precision of diagnosis.

The methods of study of compounds which have been discussed in this communication lend themselves also to attack on the other radiopaque diagnostic problems. These are attracting our intensive attention at this time. We hope before very long to be able to report to the medical public the availability of materials for visualizing the other diagnostic areas of the body in the same way as we have been able to improve the performance in the two areas discussed in this paper.

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CARCINOMA OF THE STOMACH ; SEVEN-YEAR REVIEW

*JOHN F. SANABRIA, M.D.**

A study of forty-one cases of carcinoma of the stomach treated at the Department of Surgery of the San Juan City Hospital was presented in December, 1952.[1] The present review adds thirty-six new cases, bringing the total to seventy-seven, seen between July 1, 1947 and October 4, 1954. (See Table I)

TABLE I
NUMBER OF CASES BY YEAR

1947	(second semester	2
1948		7
1949		5
1950		7
1951		10
1952		18
1953		16
1954	(until October 4)	12
	TOTAL	77

The number of patients treated for this ailment at this institution has recently increased at a greater rate than in previous years. During the last three years, forty-six patients have been subjected to operation for carcinoma of the stomach as compared with fourteen treated during a comparable period prior to 1950. This represents an increase of over 300%.

Sex incidence:

Out of seventy-seven patients, there were fifty-two males and twenty-five females (a ratio of 2:1).

Age incidence: (See Table II)

The age incidence varied from twenty-two to eighty-six years with a preponderance in the seventh decade. Fifty-six of the seventy-seven patients were between forty and seventy years of age.

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TABLE II

AGE INCIDENCE

Age in Years		No. of Patients
21 to 30		5
31 to 40		4
41 to 50		17
51 to 60		13
61 to 70		26
71 to 80		10
Over 80		2
	TOTAL	77

Presenting Symptom: (See Table III)

Epigastric distress was the most common symptom, being present in thirty-one patients (40%), with from one and a half months to eight years' duration. Epigastric pain occurred in eighteen patients (23%) and lasted from one month to five years before admission. Dysphagia was complained of by seven patients (9%), with a duration of from one to eight months. Hematemesis was the presenting complaint in four patients (5%). One patient had only one episode of bleeding, whereas in the other three recurrent episodes of bleeding had occurred for a period of one month in one, and for two years in the other two cases. Right upper quadrant mass was noticed by four patients (5%), its duration varying from three months to one year. Three patients (4%) had nausea and vomiting with a duration of two months, four months and four years respectively. The remaining ten patients had atypical presenting symptoms.

Physical Findings:

Clinical evidence of weight loss was present in forty-seven patients. An epigastric or right upper quadrant mass was palpable in twenty-four. Five had epigastric resistance to palpation. Rectal shelf was reported in four patients. Hepatomegaly was found in nine. Virchow's node and jaundice were present in one patient each.

TABLE III

PRESENTING SYMPTOM

SYMPTOM	PERCENTAGE
Epigastric distress	40.2
Epigastric pain	23.3
Dysphagia	9.1
Hematemesis	5.2
RUQ mass	5.2
Nausea and vomiting	4.0
Epigastric and RUQ pain	1.3
LUQ pain	1.3
Vomiting and diarrhea	1.3
Diarrhea and tarry stools	1.3
Anorexia and weight loss	1.3
LLQ pain radiating to epigastrium	1.3
Epigastric pain with postprandial vomiting	1.3
Epigastric distress with anorexia, nausea and vomiting	1.3
Periumbilical pain with vomiting	1.3
Epigastric fullness after meals	1.3
TOTAL	100.0%

Laboratory Data:

Hemoglobin determinations showed values below 10 gms/100 cc. in forty-two. Hemoglobin value above 12 gms./100 cc, without evidence of dehydration, was seen in twenty-two patients (28.5%).

Gastric analysis with histamine stimulation was performed in thirty-three patients. Achlorhydria was present in fifteen (45.5%), hypochlorhydria in seven (21.2%), hyperchlorhydria in three (9%) and a normal response in the remaining eight (24.3%).

Benzidine test for occult blood in gastric contents was done in thirty-three patients. It was found frankly positive in twenty (60.6%), slight traces in one (3%) and negative in twelve (36.6%).

Examination of the stool for the presence of occult blood was done in twenty patients. It was found positive in eight (40%) and negative in twelve (60%).

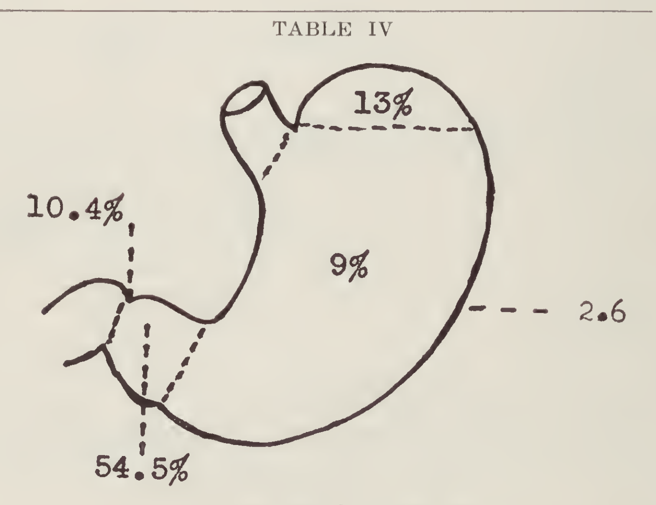
Roentgenographic Findings: (See Table IV)

Lesions involving the antrum of the stomach were demonstrated in forty-two patients (54.5%), pyloric tumors in eight (10.4%) and lesions of the body and cardia in seven (9%) and ten (13%) respectively. The growth originated in the greater curvature in only two patients.

Ten lesions involved both body and antrum. The cardia and lower esophagus were involved in four, the body and cardia in one, and the entire stomach in one. One lesion invaded the body, cardia and lower esophagus, causing partial obstruction. Another lesion was reported as a marginal ulcer. In 10.5% of the cases no films were available.

Obstruction was manifested clinically and/or radiologically in twenty of the forty-nine lesions involving the antrum and pylorus. The obstruction was partial in thirteen, complete in six and in one obstruction of the transverse colon was produced by extrinsic pressure of the tumor.

TABLE IV



Roentgenographic Findings.

Esophagoscopy:

Five of the cases diagnosed radiologically as carcinoma of the cardia were subjected to esophagoscopy. Biopsy studies were done in four of these and reported positive in all of them.

Complicating Diseases:

Secondary anemia (hemoglobin below 10 gms/100cc) was found in forty-two patients; generalized arteriosclerosis in eleven; arteriosclerotic heart disease in five; inguinal hernia in five;

benign prostatic hypertrophy in four; hookworm infestation in four; minimal pulmonary tuberculosis in three; senile emphysema in three; hydrocele in three; hypertension in two; senile cataracts in two and cirrhosis of the liver in two. The following diseases were found in one patient each: ascariasis, congestive heart failure, hypertensive cardiovascular disease, syphilis, chronic bronchitis, bronchial asthma, severe jaundice, rectal polyp and chronic osteomyelitis of the frontal bone.

Unresected Cases: (See Table V)

Of the seventy-seven patients with carcinoma of the stomach, twenty-seven had surgical exploration without resection of the tumor and one had needle biopsy of the liver for a total of twenty-eight unresected cases (31.1%). Of these, eighteen (64%) had a palliative procedure done. Fourteen patients had a gastro-enterostomy performed, five anterior and nine posterior. Gastrostomy was done on two patients, ileosigmoidostomy on one and esophago-jejunostomy with jejuno-jejunostomy on one.

Exploratory laparotomy alone was done on seven patients (25%) and exploratory thoracotomy on two.

Biopsy from either the primary lesion or from grossly metastatic foci was obtained in nineteen (68%) of the unresected cases. In thirteen of these it was reported positive, whereas in four it was negative in spite of the metastatic spread observed at operation. Of the thirteen specimens reported positive for cancer, five were diagnosed as adenocarcinoma, four as colloid carcinoma and four as anaplastic carcinoma.

TABLE V

CARCINOMA OF THE STOMACH

	PATIENTS	PERCENTAGE
Total cases	77	100.0
Total cases explored	76	98.7
Resected	49	63.9
Total gastrectomy	13	
Subtotal gastrectomy	36	
Unresected	28	36.1
Palliative procedure	18	

RESULTS IN UNRESECTED CASES

Mortality:

There were four deaths out of the twenty-eight unresected cases, with a mortality rate of 14.3%. One patient developed intractable diarrhea following gastrostomy with consequent electrolyte imbalance and died on the sixth post-operative day with bronchopneumonia and lung abscess. One died with acute generalized peritonitis following paracentesis done for the relief of ascites on the sixth post-operative day. Another patient developed a fistula at the site of the anastomosis and died on the twenty-ninth day after an esophagojejunostomy.

Complications:

Of the remaining twenty-four patients who survived laparotomy, one had a wound dehiscence with recovery; two developed ascites requiring repeated paracentesis; another had left pleural effusion and one developed acute glomerulonephritis, bronchopneumonia and partial dehiscence that healed.

Follow Up of Unresected Cases:

No information about the time or cause of death could be obtained in five patients and one could not be followed. Of this group there are only two patients alive, three and four months postoperatively, both bed-ridden and in terminal condition. The remainder patients died as follows: Three died two weeks postoperatively, two after one month, one after two months, three after three months and one after five, seven, nine and eleven months respectively. Of the three patients that lived over one year, two died fifteen and one eighteen months after operation.

RESECTED CASES

Gastrectomy was performed on forty-nine patients (63.9%), thirteen of which had a total removal of the organ and thirty-six a subtotal resection. The total gastrectomies included omentectomy, splenectomy and excision of regional lymph nodes. The subtotal resections were done with omentectomy in seventeen patients and with splenectomy in eight.

Distal esophagectomy was performed in six patients, partial pancreatectomy in four, partial hepatectomy in two and partial excision of the transverse colon in two.

The surgeon reported lymphatic metastasis in forty-two patients. This figure includes the thirteen patients subjected to

total gastrectomy. There was no evidence of lymphatic metastasis in seven cases. However, there were sixteen specimens reported by the pathologist as free of lymph node involvement, so that in the other nine cases inflammatory lymphadenopathy was interpreted at operation as lymphatic spread of the neoplasm.

Histopathologically the lesions were classified as follows: thirty-eight adenocarcinomas, five anaplastic carcinomas, four colloid carcinomas, two schirrous carcinomas and one adenocarcinoma of the signet ring cell type.

RESULTS IN RESECTED CASES

Mortality: (See Table VI)

Of forty-nine gastric resections there were seven deaths, with a mortality of 14.3%.

TABLE VI
DEATHS AFTER RESECTION

NAME	MAJOR COMPLICATION	DAYS POST-OP.
1— NNT	Pulmonary atelectasis	0
2— AMS	Leakage from duodenal stump	13
3— MNA	Leakage from duodenal stump	22
4— PMH	Leakage from anastomosis	27
5— RVG	Peripheral vascular collapse	12
6— FCG	Unknown	1
6— JRS	Saddle embolus	6

Post-Operative Complications:

Fourteen patients developed complications post-operatively while twenty-eight had an uneventful course. The complications were as follows: bronchopneumonia, pleurisy, thrombophlebitis, electrolyte imbalance, post-operative shock, urinary retention secondary to benign prostatic hypertrophy, laryngeal edema, hematoma of the wound, subdiaphragmatic abscess and complete obstruction at the site of anastomosis with paralytic ileus.

Follow Up of Resected Cases: (See Table VII)

The longest survival is that of M.O.A. (#98011) who had a subtotal gastrectomy in 1946 for a gastric tumor with regional metastasis. The procedure was palliative. She was well until 1952

TABLE VII

FOLLOW UP OF RESECTED CASES

No.	Name	Hosp. No.	Year	Lymphatic Metastasis	Duration
1—	MOA	98011	1946	Present	7 years
2—	AMS	19365	1947	Present	13 days
3—	CT	32123	1948	None	13 months
4—	SG	32812	1948	Present	15 months
5—	JSB	31652	1948	None	5 years
6—	EL	33562	1948	None	6 years (living)
7—	EVS	29963	1948	None	6 years (living)
8—	FR	52054	1949	None	14 months
9—	FMA	49120	1949	None	4 years
10—	FN	64174	1950	None	2 years
11—	VLR	38654	1950	Present	4 years (living)
12—	AMN	60359	1950	None	5 months
13—	NNT	78724	1951	None	Died at operation
14—	MRP	78385	1951	Present	3 years (living)
15—	JDA	76842	1951	Present	Not seen since Op.
16—	JPH	85522	1951	Present	2 years
17—	ZT	79913	1952	Present	6 months
18—	JFM	96103	1952	Present	11 months
19—	JDP	98270	1952	Present	1 year
20—	MC	99777	1952	Present	15 months
21—	JOS	21812	1952	None	2 years (living)
22—	RF	65622	1952	Present	2½ yrs. (living)
23—	MOA	98011	1952	Present	(See No. 1 above)
24—	JRS	102911	1952	Present	6 days
25—	TRC	104188	1952	Present	10 months
26—	ANA	96128	1952	None	14 months (living)
27—	JH	13129	1952	Present	2 years (living)
28—	RVG	86311	1953	Present	12 days
29—	FCG	106283	1953	None	1 day
30—	JRP	65606	1953	Present	5 months
31—	PCI	5317483	1953	Present	1 month
32—	OSL	5318403	1953	Present	8 months
33—	ADF	534671	1953	Present	20 months (living)
34—	JTG	539509	1953	Present	7 months
35—	JRR	536574	1953	Present	13 months
36—	PCT	538825	1953	Present	3 months
37—	RFC	5312124	1953	Present	15 months (living)
38—	FSF	13765	1953	Present	5 months
39—	EDL	536474	1953	Present	19 months (living)
40—	URC	106835	1953	None	9 months
41—	JSC	53284	1953	Present	22 months (living)
42—	AAF	532502	1953	Present	8 months
43—	MNA	54611	1954	Present	22 days
44—	JRD	5315994	1954	Present	10 months (living)
45—	JMR	5315136	1954	None	9 months (living)
46—	FBR	108659	1954	Present	Not seen since Op.
47—	NRS	545714	1954	Present	3 months
48—	MPA	547333	1954	Present	2 months (living)
49—	MB	547308	1954	None	5 months (living)
50—	PMH	24787	1954	None	27 days

when recurrence of the tumor was found at the site of anastomosis with infiltration of the pancreas. She was re-operated upon on April, 1952, having a resection of the tumor and remaining stomach with omentectomy, splenectomy and partial pancreatectomy. The patient died seven years after the original operation.

Of the five patients resected in 1948 there are two living and well (six years) and one lost to follow-up after the fifth year. Of the two resected in 1949 one was followed for four years and has not been located since then. Of the three patients resected in 1950 one is living and well (four years) and one could not be followed after two years. Of four patients resected in 1951 only one is living (three years). Of eleven patients operated upon in 1952 there are four living. Of fifteen patients resected in 1953 there are four living. Of eight patients resected in 1954 (until October 4) there are four living.

CONCLUSIONS

The operability rate of patients seen in our Surgical Department has been 98.7%, resectability rate 63.9% and operability for palliation 64%. Of the resected cases 26.5% had total and 73.5% subtotal gastrectomy. The mortality rate of resected and unresected cases has been 14.3% for each group, with the same percentage for over-all mortality. Those patients that could not be followed are considered as dead from cancer.

Our final results are as follows: Nine patients were operated upon five years or more (before 1950). Of these, four lived over five years, with 44.4% of five-year survival in determinate cases. Of the remaining patients nineteen lived more than one year, eight patients more than two years, three more than three years and two over four years.

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RETROLENTAL FIBROPLASIA
(Retinopathy of Prematurity)*

SOME CASES IN PUERTO RICO

GUILLERMO PICÓ, M.D.

In 1942 a new eye disease was first described by Terry[1] in premature infants. He found an opaque fibrous tissue membrane behind the lens and for this reason gave the name of retrolental fibroplasia to the condition. Terry established the relation of this disease to premature infants.

INCIDENCE

Since that first report the incidence of the condition has been rapidly increasing. In a recent paper presented by Dr. Franklin M. Foote,[2] Executive Director of the National Society for the Prevention of Blindness, it was reported that 5,000 children in the United States are blind as the result of this terrible disease. It is now the most frequent cause of blindness in infants. Reports of this disease have come from the United States, European countries and Australia. It occurs almost exclusively in premature infants weighing less than 4 pounds but Reese[3] has found that about 2% of cases occur in infants with normal birth weight. The incidence is inversely proportional with the birth weight. Some hospitals have reported an incidence as high as 80% and others of only about 10% in those premature infants. It is usually bilateral but may be unilateral. There is no difference in sex incidence.

CLINICAL PICTURE

Immediately after birth the eyes are as normal as in other premature infants who do not develop the condition. We must remember that the eyes of premature infants show several differences when compared to the eyes of adults. Tyner[4] has listed these differences as follows:

1. Haziness of the ocular media.
2. Pallor of the optic disc.
3. Enlarged retinal veins.
4. Gray color of the peripheral retina.

Szewczyk[5] believes that the last two findings are definitely pathological.

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* From the Section of Ophthalmology, School of Medicine, University of Puerto Rico. Presented at the annual Meeting of the Section of Pediatrics, Puerto Rico Medicl Association, February 5, 1955.

Patz[6] has described the presence of a preliminary stage of marked constriction of the retinal vessels in some premature infants who later developed the classical picture of retrolental fibroplasia.

The process of development of retrolental fibroplasia has been divided into two phases:

1. The active phase.
2. The cicatricial phase.

A committee for the study of retrolental fibroplasia was appointed recently by the National Society for the Prevention of Blindness. A subcommittee presided by Dr. Algernon B. Reese submitted a classification of different stages and grades of the disease under the active and cicatricial phases and it has been generally accepted by all workers in this field. It is very important that all ophthalmologists and pediatricians be familiar with this classification so as to obtain uniformity in the description of reported cases. This will help in the accurate evaluation of the course of the disease in different places and of the effect of different factors. This classification[7] is as follows:

Stages of Retrolental Fibroplasia in the Active Phase

1. Dilatation and tortuosity of retinal vessels.
   Hemorrhages may or may not be present.
   Early neovascularization especially in the extreme periphery of the visible fundus, may be present.

2. Stage 1 plus neovascularization and some peripheral retinal clouding.
   Hemorrhages are usually present.
   Vitreous clouding may or may not be present.
   Spontaneous regression may occur.

3. Stage 2 plus retinal detachment in the periphery of the fundus.
   Spontaneous regression unlikely.

4. Hemispheric or circumferential retinal detachment.
   Elevation of the retina over a large area, but still with some retina in position.

5. Complete retinal detachment.

Grades of Retrolental Fibroplasia in Cicatricial Phase.

1. Small mass of opaque tissue in periphery of the fundus without visible retinal detachment.

The fundus may have a pale appearance.
The blood vessels may be atenuated.

2. Larger mass of opaque tissue in periphery of the fundus with some localized retinal detachment.

   The disc is distorted by traction toward the side of the tissue, which is usually temporally.
   Cases ending in Grade I or II have useful vision.

3. Larger mass of opaque tissue in periphery incorporating a retinal fold which extends to the disc.

   Visual acuity varies from 5/200 to 20/50.

4. Retrolental tissue covering part of pupillary area.

   Small area of attached retina may still be visible or only a red reflex over a sector of the fundus may be seen.

5. Retrolental tissue covering entire pupillary area.
   No fundus reflex present.

The early changes in the retina are first noticed clinically about the end of the third week and consist of dilatation and tortuosity of the retinal vessels.

After the dilatation of the blood vessels, edema in the form of grayish discoloration and brush-like new blood vessels and hemorrhages appear in the periphery. Spontaneous regression may occur in this second stage. In the third stage there is retinal detachment in the periphery of the fundus which later progresses in the 4th. and 5th. stages to complete retinal detachment. In these last three stages, vascular buds arise from the retina and extend to the vitreous. This angioblastic process was shown in pathological reports by Reese and Blodi[3] and others.[8]

After the active phase comes the healing which leaves permanent damage. The active phase of the disease subsides at about three months of age and is followed by the cicatricial phase. The appearance of the cicatricial phase is divided in five different grades. These may vary, from the first grade in which a small mass of opaque tissue in the periphery of the fundus is seen together with a pale fundus and contracted blood vessels, to the advanced grades in which there is more opaque tissue in the retina and vitreous. In Grade V, the retrolental tissue covers the entire pupillary area.

In many cases the disease is first noticed by the parents when they see the opaque mass of detached retina and vascularized fibrous tissue behind the lens.

DIAGNOSIS

All premature infants should be kept under the observation of an ophthalmologist. He should do careful and regular ophthalmoscopic examinations, at least once weekly. The pupils should be widely dilated with a mydriatic, such as 10% Neosynephrine solution, to make possible a thorough examination of the eyegrounds. The examinations should start when the child is two weeks old and may be stopped at the age of three months if by that time there is no evidence of retrolental fibroplasia. Reese[3] states that retrolental fibroplasia has never been known to develop in an infant older than three months. The diagnosis is simple when the infants has thus been kept under observation. However, if the infant is first seen in the advanced stages when there is a mass in the vitreous, we must then take into consideration other possible conditions.

DIFFERENTIAL DIAGNOSIS

1. **Retinoblastoma.**

It may be very difficult to eliminate the possibility of a retinoblastoma when the infant is first seen when several months old, and has as main finding a mass behind the lens. The history of being a premature infant is of great help in favor of retrolental fibroplasia. Calcium deposits can be demonstrated by X-ray examination in a large percentage of cases of retinoblastoma. Reese[9] states that dentate processes in the periphery of the opaque retrolental mass are characteristic of retrolental fibroplasia and are not seen in cases of retinoblastoma.

2. **Persistent Hyperplastic Primary Vitreous**
(Tunica Vasculosa Lentis)

This condition resembles the appearance of the advanced stages of retrolental fibroplasia but it occurs unilaterally and in full-term infants.

3. **Pseudo-glioma.** (Metastatic Retinitis or Purulent Endophthalmitis)

Following an infectious disease in childhood an infected embolus may reach the eye and produce a purulent endophthalmitis with an abscess in the vitreous. Later on fibrosis and detachment of the retina may occur.

The history of a previous febrile disease accompanied by acute inflammation of the eyeball will give the diagnosis in these cases.

ETIOLOGY

During the last decade numerous studies have been done to determine the cause of retrolental fibroplasia. Among the etiologic possibilities that have been eliminated are infection, dietary deficiencies, estrogen deficiencies, Vitamin E deficiency, blood transfusions, effect of light and adrenal-cortical hormone deficiency.

In the last few years it has been demonstrated that the cause of retrolental fibroplasia is the excessive use of oxygen in the care of the premature infant. It is surprising that this was not considered before when we remember that the increasing incidence of this disease, in the last 10 or 15 years, has corresponded to the use of better incubators in which the infant receives prolonged oxygen therapy at high concentrations.

Szewczyk[10] and also Ryan[11] were among the first to call the attention to the possibility of the role of oxygen in the development of retrolental fibroplasia. Szewczyk reported that the early changes of retrolental fibroplasia could be reversed by treatment with high concentration of oxygen of 65 to 70% and that the changes would reappear by removing the infants from the oxygen incubators. Ryan, on the contrary, thought that the disease was due to the administration of an excess of oxygen. Campbell[12] in Australia and Crosse and Evans[13] in England showed that it occurs in cases receiving excessive oxygen. Crosse and Evans reported that when oxygen was restricted the disease did not occur in 65 infants examined.

Patz and co-workers[6-8] have done and excellent work, both experimentally with rats, mice, kittens and puppies and with clinical patients, to demonstrate the effect of an excess of oxygen in the immature retina. They have obtained in the animals the typical changes of retrolental fibroplasia by exposing them to 60 to 80% oxygen concentrations. They have correlated these findings with controlled studies in clinical patients. On an alternate admission basis, the premature infants were placed into either a high oxygen group or a low oxygen group. The infants in the first group received 60 to 70% constant oxygen concentrations while those in the second group received oxygen concentrations under 40% and only for specific clinical indications. Twelve out of 60 infants in the high oxygen group (20%) developed advanced retrolental fibroplasia while only one out of 60 patients in the low oxygen group developed the advanced disease. No significant difference in mortality rate was noted between the infants in the high and in the low oxygen groups. It was observed during the study that 21 cases of active disease (Active stages 1 and 2) regressed to normal in the high oxygen groups and 9 cases

of active disease regressed to normal in the low oxygen group. From these findings we may see that nearly 60% of the infants in the high oxygen developed some stage of retrolental fibroplasia although many of those had spontaneous regression to normal. In low oxygen only about 15% of the infants developed some stage of retrolental fibroplasia but all except one had spontaneous regression to normal. Similar work with similar results was also published recently by Lanman and his co-workers.[14] Both Patz and Lanman have indicated that the therapeutic use of oxygen for the treatment of early retrolental fibroplasia, as suggested by Szewczyk, should not be used. They remind us of the high spontaneous regression rate in retrolental fibroplasia and that this regression has been detected in some cases that were still in incubators with high oxygen. The disease has also been seen to appear while the infants are still in the incubator. This are very strong arguments against Szewczyk's theory that retrolental fibroplasia was produced by too rapid withdrawal of the infant from the oxygen.

PATHOGENESIS OF RETROLENTAL FIBROPLASIA

Patz[8] has described the condition of the retinal vasculature and its relation to oxygen as follows:

1. In premature infants, the peripheral retinal vascularization is not completely developed.
2. Oxygen saturation of arterial blood in fetus is approximately 50%. After birth is 90%.
3. The principal stimulus for growth of immature retinal vasculature is a relative oxygen want - hypoxia - in the retinal periphery.
4. The final maturation of the vasculature of the retina of premature infants occurs at arterial oxygen concentration of 90% where in normal gestation the fetus would have continued to develop in utero at considerably lower oxygen tension. This increase of oxygen in the arterial blood of the prematurely born infant may bring changes of retrolental fibroplasia even without the use of incubator oxygen.

Patz[8] describes the effects of oxygen on the retina with an immature vasculature into two phases:

Oxygen ---➤ elevates choroidal oxygen tension ---➤ increased diffusion across the retina eliminates normal anoxic growth stimulus ---➤ retinal vessel growth suppressed and the become attenuated or obliterated (Vasoconstrictive phase) ---➤ when growth is finally activated the normal channels of vascularization probably no longer exist and blood vessels then erupt into vitreous and vasodilation and other changes occur. (Proliferative phase).

TREATMENT AND PREVENTION

Retrolental fibroplasia is a disease which may have spontaneous regression but apparently there is no specific treatment that may change the course of the disease. Preventive measures consist in the use of oxygen in very limited concentrations, less than 40%, for short periods, and only for the treatment of cyanosis. Patz[8] and also Lanman[14] found that in their separate similar controlled studies the mortality rate in the premature infants was not influenced by the amount of oxygen that was used. They insist that the pediatricians should avoid the abuse of oxygen. It is very important that an oxygen analyzer be used to measure accurately the oxygen concentration in the incubators.

RETROLENTAL FIBROPLASIA IN PUERTO RICO

I am sure that you are interested to know if retrolental fibroplasia occurs in Puerto Rico. I have seen two cases which showed in the eyegrounds typical cicatricial bands of fibrous tissue as are seen in stage 3 of the cicatricial phase of retrolental fibroplasia. One of these children was a premature child which was born at the mother's home and did not receive any oxygen in the neonatal period. He was 10 years old when I first saw him. The cicatrical band started in an area of scar tissue that involved the region between the disc and the macula and it extended anteriorly into the vitreous towards the ora serrata.

The other child had a similar pathology but he was born at term and apparently with normal weight. This child also did not receive any oxygen therapy after birth.

The number of cases reported in the literature of the development of retrolental fibroplasia in an infant that has not received supplementary oxygen are very small. Some authors[14-15] have suggested that there might be other etiological factors aside from oxygen which may also affect the retina and produce the changes of retrolental fibroplasia. The sudden increase of oxygen from 50% in the arterial blood in the fetus to 90% at birth may be the cause of retrolental fibroplasia in premature or full-term infants who have not received incubator oxygen, according to the hypothesis of Patz.[8]

I have seen another infant, 8 months old, who showed even more typical findings of retrolental fibroplasia. We were unable to obtain a complete and accurate history from the relatives aside from the fact that the child was born prematurely, at the parents home, and did not receive any oxygen after birth. The right eye showed almost total retinal detachment and new blood vessels were

seen in the detached retina. From the periphery of the retina bands of fibrous tissue extended anteriorly through the vitreous toward the lens. The left eye showed retinal detachment in the periphery below, and in that area were seen numerous hemorrhages and newly formed blood vessels. The retinal veins were very dilated and tortuous as they approached the area of retinal detachment. The rest of the fundus had patches of retinal edema and scars. This child was apparently blind and the pupils were dilated and did not react to light.

I have not heard of other cases reported in Puerto Rico and as far as I have been able to find there are no other cases reported in the literature from other Latin American countries. Apparently the Pediatricians in Puerto Rico have been conservative in the use of oxygen in the premature infants and the available incubators have not been of the ideal types. This must be the explanation for the almost total absence of retrolental fibroplasia here and we have been very lucky.

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TRAUMATIC PARAPLEGIA*

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When we now see paraplegia patients who, though confined to wheelchairs, are apparently in good physical health and actively engaged in an occupation, many of us will recall patients from the pre-World War II era who were not nearly so fortunate. These nightmare cases gradually developed decubitus ulcers covering a large portion of the body. Considered hopeless, they became dope addicts. They developed bladder and kidney stones, urinary tract infections, wasted away to a mere skeleton, and when death came after one or two years, it was welcomed by patient, doctor, family, and all concerned.

We saw a considerable change during World War II when over 80 per cent of the spinal cord injuries survived the initial stages of their illness and were collected in large Army centers. Here, they were treated by representatives of a number of specialties who, through close collaboration, brought about considerable improvement in their treatment. Such treatment is a little more difficult for the individual traumatologist who has the total care of such a patient. However, he can do considerable provided he is cognizant of the possibilities.

In reviewing these problems, we will take them up according to the specialties in which they seem to occur.

First of all, on admission the patient confronts us with a lesion involving the vertebral column and spinal cord. It is well for the orthopedist and neurosurgeon to collaborate on evaluating this situation; however, the nerve injury takes precedence and in many places the neurosurgeon is in charge of the immediate treatment. The problem then is whether to do an immediate laminectomy on this very sick patient who can't stand much additional trauma or to delay the procedure until one week allowing edema to subside. Most feel that an immediate laminectomy within the first 12 hours is indicated if there is complete loss of function, spinal fluid block, or x-ray evidence of laminal fracture providing the patient's condition and operative facilities permit. Otherwise, the procedure is postponed until one week. However, late laminectomy after two weeks is of little value. Whether or not a laminectomy is decided upon, the patient is placed in skeletal traction employing Crutchfield tongs or the equivalent in all except the mildest cervical lesions. Head-halter traction has little place in such treatment since insufficient weight can be ap-

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plied without the development of ulcers. Spine fusion is contraindicated at the time of immediate laminectomy for several reasons. First, it is unwise to prolong the operation on a patient in initial shock when it can be performed safely later. Secondly, a vascular bed formed by interrupting many bony capillaries must be prepared for an adequate fusion, leading to the formation of a hematoma which further threatens the acutely traumatized cord. Thirdly, it is difficult to determine at this stage whether a fusion will be necessary or not. This procedure can be combined with laminectomy in the occasional case in which a late laminectomy is thought to be of value such as the partial lesion in which there has been some loss of function through bony impingement, scar tissue constriction, or in some cauda equina lesions where the response is more like that of the peripheral nerve injury.

A serious complication which must be combated right from the start are decubitus ulcers, falling in the sphere of the plastic surgeon. It would seem unnecessary to mention that a patient must be turned to avoid pressure over bony prominences, but it is surprising how many patients one sees who have been allowed to lie flat on their backs for two to three weeks while supposedly treated for their skeletal lesions. Decubitus ulcers are easier prevented than cured, and it is our practice to place the patient on a Stryker frame or Foster bed immediately on admissión. The patient is them turned every two hours in the day and four hours at night. Care must also be taken that the skin is kept clean and dry with prompt attention to urinary spillage and fecal contamination. Inadequate tretment leads to ulcer formation over the bony prominences of the heels, knees, trochanters, ischial tuberosities, ilium or sacrum.

Protective dressings, padding the bony prominences have proved helpful in their prevention, particularly in the emaciated individual. Application of these and ulcer dressings is facilitated by the use of an electric hoist so that the patient need not be lifted with every turn of the bandage. Abdominal pads and bias-cut stockinet have proved useful for this purpose.

Superficial ulcers may heal spontaneously with proper care as the patient's condition improves. In general, ulcers over 3 cm. in depth or 5 cm. in diameter must be closed surgically, as recommended by Freeman[4]. In so doing, the lining of the cavity which often undermines the margin for a considerable distance is carefully excised. The underlying bony prominence is resected and the defect closed by advancing or rotating flaps from areas subject to less pressure. In resecting the greater trochanter, it is well not to remove more than half the distance to the lesser trochanter or fracture may result. Large ulcers in the posterior trochanteric regions may require a femur shortening to obtain enough soft

tissue for closure. If the hip joint is extensively infected through continuity with the trochanteric ulcer, a resection of the joints may be attempted with some hope of success. After two such attempts, however, amputation by hip disarticulation may be necessary.

In performing a hip disarticulation, it may be necessary to vary the usual routine depending on the size and location of the ulcers. If the trochanteric ulcer is not too large or is anteriorly placed and if an ulcer is also present over the anterior superior spine, the usual large posterior flap is quite satisfactory. However, if the trochanteric ulcer lies more posteriorly and particularly if in connection with an ischial ulcer, the posterior flap cannot be obtained and an anteromedial flap is utilized. In some patients, neither flap is obtainable and it is necessary to do a two-incision amputation. This is first accomplished at a lower level through the upper thigh region and the stump filleted, saving the circulation from the lateral and medial circumflex vessels. The ulcer defect is then closed as a separate incision.

Another problem which may lead to amputation and which taxes the ingenuity of the orthopedist is that of myositis ossificans or the excessive bone formation which occurs in the region of joints, particularly around the hip and less frequently at the knee. This may effectively bridge the joint like an extra-articular arthrodesis. Resection alone is seldom sufficient due to a prompt recurrence of the bone formation. A pseudo-arthrosis procedure, such as a Jones pseudo-arthrosis, will often give satisfactory results; however, even this may again become solid. Results have proved unsatisfactory even after excising a 2-inch segment of the femur, carefully closing muscle between the bone ends with approximated fascial surfaces, and amputation has eventually been necessary. There have been many proposed theories regarding the cause for this bone formation, but it has still remained an unsolved mystery. It is more common in the colored race who on the other hand are rarely bothered by kidney stones.

The role of the urologist in the treatment of paraplegia is a very important one since care of the genito-urinary tract to avoid complications must begin immediately on admission. Even with the aid of our present-day antibiotics, urinary tract infection is still a common cause of death in these patients. During the early atonic stage, some form of bladder drainage is necessary and during World War II it became almost routine to perform a suprapubic cystostomy. This is now considered by most to be unnecessary even for the quadriplegics who cannot aid the emptying of the bladder by manual pressure. A catheter should be inserted immediately on admission. The Foley type is of considerable help

and a No. 14 has been found to be a satisfactory size. This should be changed every two days to prevent infection. Tidal drainage is considered preferable to free drainage since it more nearly approximates the normal bladder function. This, as designed by Dr. Monroe, provides for alternate filling and emptying of the bladder and may be used to follow the progress of the bladder by cystometrogram determinations. At about four to six weeks following injury, many patients develop an automatic bladder and bladder training becomes quite important. The normal balance of the forces of emptying and retention is disturbed in these patients and may result either in a small contracted bladder or a distended bladder with marked residual urine. The contracted bladders may often be helped by selective sacral rhizotomy, pudendal neurectomy, or other means of completing the denervation of the bladder. Bladders which fail to empty are often helped by removing obstruction in the region of the vesical neck.

The catheter which was begun on admission is removed as soon as possible since it is in itself a foreign body tending toward infection. When functioning automatically, the catheter is removed and the bladder checked for residual urine. If it does not exceed 60 c.c., the patient can generally do without the catheter. It is then necessary to provide some means for collecting the urine. A clamp placed on the penis and removed at regular intervals has been found to be satisfactory by some; however, the pressure must be carefully adjusted to avoid ulcer formation. Somewhat more satisfactory is to tape a rubber condom to the penis connected by a tube to the jar or leg urinal.

The chief complications which may result in the course of the patient's care are urinary tract infection, stone formation, epididymitis, and urethral fistula. Ascending infection is avoided by proper drainage of the kidney and relief of residual and ureteral reflux. Drainage is improved when the patient can be gotten up early in a wheelchair or ambulating with crutches which also help to stabilize the calcium in the bones, cutting down on stone formation. In treating infection with antibiotics, it is well to be careful to avoid diarrhea as sometimes occurs with aureomycin and achromycin. Gantrisin and furadantin have been found to be very useful for the gram-negative organism. Large renal calculi and free stones blocking the ureter are removed surgically. Bladder stones are removed by lithotripsy. Urethral fistula and epididymitis are rare when a catheter of the proper size is used, changed frequently, and removed as soon as the patient can do without it.

As soon as the patient's condition will permit, it is desirable to get him upright to drain the kidneys, stabilize calcium in bones,

and aid him in rehabilitation. It is often difficult to go immediately from a recumbent to an upright position without the patient's blacking out due to pooling of blood in the large abdominal vessels. An abdominal support is of help. It is also possible to get the patient up gradually using an electric hoist connected to one end of the Foster bed or by tilting the patient on an operating room table. Still another method which has been tried is to place brackets on the end of a Stryker frame and stand the patient up sandwich between the two halves of the frame, thereby supporting both the abdomen and the spine. Most patients who have escaped complications can be gotten up in the wheelchair at about six weeks following injury. At this early date, some form of spine support is generally necessary to support their bony lesions. The usual leather and metal braces which are easily adjusted have been found quite satisfactory for this purpose using a Knight type for lumbar lesions, Taylor brace for the lower dorsal region, and long cervical for the upper dorsal and cervical spine. After six more weeks, it is usual for there to be sufficient bony healing for the patient to go without a brace. If the site of injury is still unstable, a spine fusion may be indicated. Occasionally, a patient with an unstable cervical lesion may require bracing for a longer period of time when a Plexiglas brace may be advantageous since it can be worn more easily under ordinary clothing and is less conspicuous.

During World War II, we considered it necessary to brace the entire paralyzed portion of the spine and lower extremities. A spine brace was therefore coupled to long-leg braces at the hip which proved to be a cumbersome outfit difficult for the patient to put on and if it fitted when the patient was in the standing position, it would seldom fit while sitting. We then shifted to a separate brace for the spine and lower extremities while in some clinics the spine brace was whittled down to a pelvic band or pelvic butterflies, and now even have been largely discarded. For the leg braces, we at first used flat steel or aluminum uprights with ring locks at the knee. These were not satisfactory for two reasons. They required a lock on both uprights or too much play developed at the knee joint. This meant the use of both hands to unlock each brace. Secondly, it was necessary to reach down to knee level which required flexing at the hips. Cam locks with bail release while mechanically better were also difficult to reach and may release unexpectedly if the patient backs into some object or bumps the legs together. Laced leather cuffs above and below the knee were first employed, but have been found to be unnecessary and time-consuming to apply and remove. The most satisfactory brace that we have used to date consists of two posterior bands

above and below the knee with one strap at the top and a knee-cap in front of the patella. The pin type of knee lock activated by a lever partway up the thigh is convenient to operate and sufficiently rigid that one lock is adequate. Most patients require a foot-drop support. The Pope type ankle joint with built-in springs adjusted by a set screw is adequate for all except the most severe spasm when a fixed ankle joint must be considered. Coil spring foot-drop braces have proved convenient for the patient while in a wheelchair; however, have been subject to considerable breakage.

To fuse or not to fuse, that is one question confronting the orthopedic surgeon. Fusion is performed for three primary reasons. First, in a partial cord lesion with returning function to prevent loss of function through increased bony displacement or angulation. If a laminectomy has been performed, this is a matter of less concern. Secondly, we may fuse to relieve pain from pressure on nerve roots through arthritic changes, etc., more commonly seen in the cervical region where there is greater range of motion. Thirdly, fusion may be performed to prevent increase of deformity when patient changes from recumbent to upright position in which case it may allow the patient to become brace-free. Extensive displacement with stripping of soft and fragmentation may also fuse spontaneously. In general, a dislocation without fragmentation may prove to be unstable; however, there are exceptions to this rule. While cervical fusion is easier when laminectomy has not been performed, it can still be accomplished quite satisfactorily even after laminectomy by the use of bilateral grafts, or equally good, a prop or H-graft held by figure-of-8 wire suture. Contrary to the experience in the lumbar spine, bank bone has proved quite satisfactory in the cervical region, possibly due to a richer blood supply or less mechanical strain.

While medical and surgical treatment is in progress, patients are also treated with the aim of rehabilitating to the fullest extent of their capacity. This is easier to accomplish in a large medical center where specially trained personnel can carry the patient up through a graded program as much as he is able to master under the direction of the physiatrist. However, much can still be done in the small clinic or even at home. The speed and extent of the patient's adjustments depend upon his physical condition, determination, intelligence, and the level of the lesion. The more mature individual who has already engaged in an occupation will generally make the adjustment more readily. The attitude of the family is very important since they must not be too pessimistic or oversolicitous. They should be told at the onset that the prospect is good for the patient to become self-sufficient and sup-

port himself in a gainful occupation. The rehabilitation program requires about six months.

Rehabilitation of the patient with a cervical lesion or quadriplegic is more difficult and less complete although much can often be done to improve the function of the hands. Those in whom the hand has been allowed to stiffen during the early stages may be relieved by capsulectomy. Others with good wrist motors but lacking in finger function may be helped by wrist fusion and tendon transplant. A certain group of patients are seen in whom only a biceps and wrist extensors are present. Here, the grasps may be improved by a tenodesis anchoring the finger to the volar surface of the wrist so that when he extended the wrist, the fingers will close. Still other patients lacking this degree of function may be helped by splints to which are attached various utensils such as fork and spoon, razor, pencil, etc. A prosthetic hook activated by a table attached to an axillary loop may also be of considerable help.

As soon as the patient is able to be up in a wheelchair, he is taught how to get from his bed to the wheelchair, from wheelchair to chairs of various types, from wheelchair to bathtub, to commode, etc. Occupational Therapy is carried on to better advantage in its department after having been started on the wards. A psychological evaluation is of considerable help in planning for a more sedentary occupation. Business courses given in educational therapy may help him determine what line of work will be suitable. These patients are taught how to drive cars, how to get themselves into the car and pull the wheelchair in after them.

Ambulation is taught early since while the wheelchair remains their chief means of locomotion, ambulation is a valuable form of exercise. They are taught first how to fall since fear of falling is a deterrent to learning ambulation. They are taught several gaits, the simplest being the swing-to, starting from a position about two feet in back of the crutches and sliding the feet up to a point almost opposite the crutches. The swing-through carries the feet ahead of the crutches and is a rapid mode of ambulation. The four-point gait requires more pelvic stability and is not available to many patients with high lesions. In Manual Arts Therapy we see a patient using a standing device to increase their standing tolerance. This consists of a girdle attached around the patient's trunk which plugs into a bracket on the workbench and allows him the free use of both hands for his occupation. As development in walking improves, they learn to navigate bus steps, stairs, swinging doors, revolving doors, and as the patient leaves the hospital provided he has been carefully trained to avoid complications, he may continue to open doors into all phases of regular living.

NOTA NECROLOGICA

DR. FEDERICO VELAZQUEZ

Nacido en Santiago de los Caballeros, República Dominicana, el 28 de agosto de 1903, ha muerto en San Juan, el 18 de junio de 1955, este buen médico y cumplido caballero, que aquí desplegó los años todos de su práctica médica.

Cursó los estudios del Bachillerato en Ciencias en Santo Domingo, y los de Medicina en la Sorbona, graduándose en el 1928. A la usanza de Europa, no adquirió el grado de Doctor hasta el 1934, cuando presentó una tesis sobre la enfermedad de Nicolás-Favre, mejor conocida ahora como el linfogranuloma inguinal. Es ésta una enfermedad universal, pero fué estudiada a fondo por primera vez en países cálidos, y su selección como tema señala, desde el comienzo de su carrera, un interés particular por las dolencias de nuestros países.

Otras intranquilidades intelectuales tenía ya para aquella época el Doctor Velázquez, pues aún antes que el doctorado recibió varios Diplomas y una Medalla de Plata, concedidos por el Gobierno de Francia en virtud de diversos inventos, algunos de los cuales se patentaron en Francia, Alemania e Inglaterra. El más notable de éstos fué una ingeniosa máquina de cambio automático de monedas de distintas denominaciones.

Casado en París en el 1932, llegó a Puerto Rico en el 1934, revalidando cuatro años más tarde. Comenzó sus actividades médicas en la Clínica Neurológica del Doctor Juliá, pero atraído por las enfermedades del pecho, poco después pasó al Sanatorio Antituberculoso Insular, con el cual estuvo relacionado durante algunos años.

En el 1943 nuestra Asociación le concedió un Premio de la Legislatura Insular por el mejor trabajo científico original realizado durante ese año: "Un nuevo método para el estudio de la cinemática de la sombra cardiovascular". Ese trabajo señaló de nuevo el talento extraordinario del Doctor Velázquez para los problemas matemáticos, físicos y fisiológicos. Ya desde los años de 1928 a 1934 había realizado nuestro colega investigaciones en importantes laboratorios de París sobre la capacidad funcional del miocardio. Fué éste un tema de persistente atractivo para él, y en el cual pudo profundizar aún más cuando le becó la Universidad de Puerto Rico, del 1944 al 1946, para llevar a cabo estudios avan-

zados en fisiología cardiovascular en los Departamentos de Fisiología de la Universidad de Chicago, y del Colegio de Médicos y Cirujanos, en la "Columbia University" de Nueva York.

Como resultado de estas últimas investigaciones, desgraciadamente inéditas, inventó el Doctor Velázquez un aparato registrador de la tensión arterial, de gran precisión, y que permitía una gravación esfigmográfica automática. Esta fase se llevó a cabo en nuestra Universidad, en el Laboratorio de Física del Profesor Amador Cobas, quien fué su consejero científico y uno de sus amigos íntimos.

Ocupó, además, diversos cargos en el Departamento de Salud y en el Fondo del Seguro del Estado, y a su muerte era Médico Consultor de la Comisión Industrial. También formaba parte de la Asociación Médica de Puerto Rico, y del Capítulo local de la Asociación Americana de Especialistas del Tórax.

En el año de 1950 el Gobierno de Francia le distinguió, por sus méritos personales, elevándole a la categoría de Caballero de la Legión de Honor.

De mente y actividades polifacéticas, alternaba el Doctor Velázquez las elegancias del salón con las exigencias de la práctica médica, con los estudios científicos, y con su amor por la literatura y la pintura. Su hablar, siempre ameno y culto, estaba salpicado de comentarios clarísimos, a menudo de una lógica incisiva. Fué tan atildado en la frase como en el vestir, tan rancio por el abolengo como moderno por la visión, y tan devoto del solar patrio como de Francia, donde maduró, y de Puerto Rico, donde fructificó y rindió, prematuramente, la existencia. Los breves días de esa vida fueron ejemplo de inquietud científica, de devoción a su esposa, hermanos y amigos, de actividad cultural, y de dedicación al ejercicio de la medicina.

La Asociación Médica de Puerto Rico, al rendirle al Doctor Federico Velázquez su respetuoso homenaje póstumo, le extiende su más sentido pésame a su esposa, Doña Marie Louise Alrivy viuda de Velázquez, a su hermano mayor, el Lcdo. Don Guaroa Velázquez, Catedrático de Derecho en nuestra Universidad, y a la esposa de éste, Doña Digna Soler de Velázquez, así como a su hermano menor, el Doctor Don Rafael Velázquez.

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LEVOPHED IN CORONARY OCCLUSION

*MAURICE L. TAINTER, M.D.**

Rationale for the Current Therapy of Myocardial Infarction

The main problem in myocardial infarction is to provide sufficient oxygen for the basal metabolism of the anoxemic muscle in the infarcted area. Since oxygen supplies the energy for performing work, decreasing the work of the heart will make more oxygen available for basal metabolism providing the oxygen supply is the same. Increasing cardiac efficiency will do the same thing since this means that the same amount of work is performed with less oxygen. More oxygen can also be made available to the anoxemic muscle by increasing the coronary blood flow and by preventing the occlusion of any additional blood vessels.

The methods in use today to achieve this increased oxygenation are as follows:

1. Bed Rest and Demerol — to decrease the work of the heart.

Bed rest does not necessarily limit the patient to the supine position. Actually respiration is easier and requires less work when in the sitting position. Because of the increased energy expenditure required in the use of a bed pan and because of the danger of thrombo-embolic accidents, some people recommend the use of a chair.[15] However, rest in a chair must be strictly maintained in much the same manner as if the patient were confined to bed. Demerol relieves pain and anxiety which in turn decreases cardiac work by preventing aimless restless motions and decreasing the tonus of the skeletal muscles.

2. Oxygen — to increase arterial oxygen saturation.

Oxygen may be given by tent, mask, or intranasal catheter. In well persons, increasing the oxygen tension of the alveolar air

* Sterling-Winthrop Research Institute, Rensselaer, N. Y.

by inhaling pure oxygen only slightly increases the oxygen content of the blood. Hemoglobin carries about 99% of the available oxygen and it is 96% saturated under normal conditions, with only 1% of the oxygen in the dissolved state. Doubling the alveolar oxygen tension would increase the total oxygen carrying capacity of the blood by not more than 5%. When there is any pulmonary edema, either frank (with rales) or incipient (interstitial), the rate of diffusion of oxygen across the pulmonary and capillary epithelium into the blood may seriously limit the oxygen saturation of the hemoglobin, and doubling the alveolar oxygen tension will double the rate of diffusion, which may be sufficient to relieve the deficit.

Demerol or other sedatives may also serve to increase arterial oxygen saturation when pulmonary edema is present since they reduce oxygen demand and air hunger.

3. Digitalis and/or Quinidine — to increase cardiac efficiency, slow the heart and prevent arrhythmias.

It is well known that digitalis, although it does not increase the strength of contraction of the normal heart muscle, does influence favorably the failing heart. The positive inotropic effect of digitalis medication means that more work is performed with less oxygen consumption,[16] thus providing more oxygen for the infarcted muscle.

Obviously digitalis is not indicated unless there is clear evidence of congestive failure. Large doses of digitalis may lead to ventricular arrhythmia and should be avoided.

Failing hearts (with a normal sinus rhythm) are slowed by digitalis through a reflex vagal action due to the relief of failure.[17] Digitalis also slows the heart in auricular fibrillation by prolonging the A-V conduction time. This slowing of the heart through either mechanism produces increased coronary blood flow, as will be noted later, if blood pressure is maintained. In addition, vagal slowing causes increased cardiac efficiency.[23-24] A reduction in rate of the failing heart without increasing the cardiac stroke output would put the patient into shock and decrease coronary flow.

Correction of arrhythmias is most important since the maximum efficiency, i.e., the most work for the least oxygen consumption, is obtained with a normal sinus rhythm. Quinidine, procaine amide and Levophed are useful for this purpose. Levophed may be used either alone or with quinidine and procaine amide to counteract the fall of blood pressure produced by the latter two drugs.

4. Anticoagulants — to prevent vascular occlusion.

The primary effect of anticoagulants is to prevent thrombo-

embolic phenomena. However, following an infarct there is a great tendency for the damaged myocardium to release blood coagulating factors such as thromboplastin into all vessels supplying the periphery of the infarct. This often causes clotting in these vessels and a further extension of the infarct. This can be prevented by the use of anticoagulants such as heparin or dicoumarol. The overall mortality from myocardial infarction, in a carefully controlled study of 1,031 cases, was reduced by anticoagulants from 23.4% to 16.0%.[18] The presence of shock, which would further decrease coronary blood flow, increases the probability of coagulation in the stagnant areas. Patients with a good prognosis need not receive anticoagulants since this type of therapy is very difficult to control accurately and has its own inherent dangers.

5. Intra-arterial Blood Transfusion.

This is a controversial subject. Some authors claim beneficial results, others deny it. The rationale for intra-arterial transfusion is as follows: In myocardial infarction, shock results from the inability of the heart to contract with sufficient force to maintain blood pressure. Hence, if one perfuses the coronary circulation by means of retrograde intra-arterial transfusion, circulation to the vital coronary vascular bed may be maintained.

However, myocardial infarction does not involve blood loss, either externally or into the tissues. An overloaded vascular bed may induce pulmonary edema and failure. If shock results from insufficient cardiac output, vasoconstrictors may be used to shunt this limited output to the vital areas. If shock in myocardial infarction is due to peripheral vasodilation, it may be more rationally corrected by peripheral vasoconstriction than by trying to fill up the dilated system with blood and/or plasma. These traditional methods of treating infarction fail in a very large proportion of cases where severe shock intervenes. Almost all patients with this degree of shock (See Table 2) have died heretofore in spite of the best care available.

Rationale for the Use of Levophed in Coronary Occlusion with Shock

The recent recognition that levo-arterenol, the second hormone of the adrenal medulla and sympathetic nervous system, may play a special therapeutic role in coronary occlusion has radically improved the therapeutic prognosis. Many patients' lives are now being saved when formerly they would have succumbed without the help of this new medicament.

CHEMISTRY

Levophed(R), also referred to as l-arterenol, l-norepinephrine, or l-noradrenaline, is a primary amine which differs from l-epinephrine by the absence of a methyl group on the nitrogen.

```
            OH
            |
           / \
          /   \ OH
         |     |
         |     |- H
          \   /
           \ /      H
            |      /
      HO--C---C--N
          |   |    \
          H   H     H
```

Although l-epinephrine and l-arterenol differ only slightly in their chemical structure, there are important differences in their physiological effects.

NATURAL OCCURRENCE AND HUMAN PHARMACOLOGY

The Sympathetic nervous system and the adrenal medulla contain both l-epinephrine and l-arterenol (l-norepinephrine). The predominant pressor substance of the adult adrenal medulla is epinephrine,[1-6] whereas l-arterenol accounts for most of the pressor activity of sympathetic (adrenergic) nerve tissue.[7-8] Extracts of the sympathetic nervous chain in man have been shown to contain 91-95 per cent of their pressor amine content as l-arterenol.[8]

Numerous investigations have shown that arterenol is liberated upon stimulation of sympathetic nerves.[9-12] This substance is clearly more effective in elevating blood pressure in man than is epinephrine.[13-14] The major differences in the physiological response to these two sympathetic ergones is summarized in Table 1. These differences have led v.Euler to suggest that arterenol is the sympathetic or adrenergic nerve mediator for "routine" functions connected with regulation of circulation, in contrast to epinephrine which is the "emergency" adrenergic hormone involved in stressful situations.[8] Homeostatic adjustments do not require the marked metabolic changes or extensive cardiovascular adjustments which characterize emotional or stress states.

Rationale for Use in Coronary Occlusion with Shock

A.—**Levophed Increases the Blood Pressure** and thereby increases coronary blood flow. If the cross sectional diameter of the blood vessels remains constant, as is the case in the rigid vessels

TABLE 1

Comparison of the Effects of a Continuous Infusion of Levophed(R) with Those of Epinephrine

CIRCULATORY		
Blood pressure	Levophed(R)	Epinephrine
systolic	Increased	Increased
diastolic	Increased	Decreased
Pulse rate	Decreased	Increased
Cardiac output	Little change or decreased	Increased
Peripheral resistance	Increased	Decreased
Coronary blood flow	Increased	Usually increased
Renal blood flow	Decreased	Decreased
Cerebral blood flow	Decreased	Increased
Splanchnic Vascular resistance	Increased	Decreased
METABOLIC		
Basal metabolic rate	Unchanged	Increased
Myocardial oxygen consumption	Increased	Increased
Cerebral oxygen consumption	Unchanged	Increased
Blood glucosa	Slightly increased	Greatly increased
OTHER EFFECTS		
Central nervous system	No effect	Increased
Eosincpenic response	None	Marked

of atherosclerosis, an increase in pressure will directly increase blood flow according to the formula

$$F = P \frac{\pi R^4}{8 n l}$$

where
F = flow
P = pressure
R = radius
l = length
n = coefficient of viscosity of blood

This formula, used by engineers and physicists for calculating the flow of fluids in pipes, was developed by Poiseuille, the anatomist, for estimating blood flow in capillaries.[19]

An increased blood pressure requires an increased output of energy by the heart which is directly proportional to the pressure, since work per minute is equal to the pressure multiplied by the

cardiac output. At zero pressure, as in shock, the heart still has a very appreciable oxygen consumption. As the blood pressure is increased coronary flow increases and, even though the work of the heart increases, there is a decrease in the oxygen deficit in the myocardium. If the pressure goes too high, there may again be an increase in oxygen deficit.[20] Hence, up to the physiological limit increasing blood pressure increases the oxygen available to the myocardium.

B.—**Levophed Reflexly Decreases the Heart Rate.** This serves to increase coronary blood flow and reduces myocardial oxygen consumption. Flow into the coronary arteries occurs primarily during diastole whereas flow out of the coronary veins occurs during systole. Thus, the coronary bed is alternately expanding and contracting during each cardiac cycle. The net effect is to make it seem as if the myocardium exerted a pumping action. The relative importance of the flowing in during diastole and pumping out during systole, has not been determined for the normal human heart.[21] The diastolic inflow effect results in an increase in coronary flow when there is a longer diastole from a slower heart rate. In atherosclerosis the limiting factor restricting coronary blood flow is often the diameter of a narrowed vessel on the arterial side; hence, the duration of inflow is an important factor. Under these conditions any slowing of the heart increases the overall coronary blood flow.

Decreasing the heart rate by vagal stimulation increases cardiac efficiency. When the heart is slowed, even though cardiac output, arterial pressure and venous pressure are maintained constant, less oxygen is consumed per minute. A decrease of 20 beats per minute in the dog heart-lung preparation results in 9% less oxygen consumption per minute when the total flow is unaltered.[22]

Vagal stimulation may produce even more dramatic results, i.e., a decrease in the resting oxygen consumption of the myocardium in addition to the rate effect. This is in contra-distinction to accelerator nerve stimulation or epinephrine which increase resting oxygen consumption.[23-24] Since the rise in pressure from Levophed causes a reflex vagal slowing of the heart, one would expect the efficiency of the heart to be considerably increased by this drug. This increased efficiency results in more work for less oxygen and thus more oxygen is available for reoxygenating the anoxic areas of the damaged myocardium. Even in the normal dog, the reflex slowing of the heart following levo-arterenol increases the oxygen saturation of the venous blood in the coronary vessels by as much as 34%.[25]

C.—**Levophed is a Direct Coronary Dilator** and thus helps to increase coronary blood flow. That there is an increase in coronary flow following Levophed is undisputed. Whether or not this increase is due in some measure to a direct coronary vasodilator effect or is secondary to metabolic effects has been questioned.[26] Studies of this phenomenon in the patient would obviously be extremely difficult. However, it has been demonstrated in the isolated perfused pig coronary artery that Levophed has a direct dilating action, independent of heart rate, metabolic effects and blood pressure.[27]

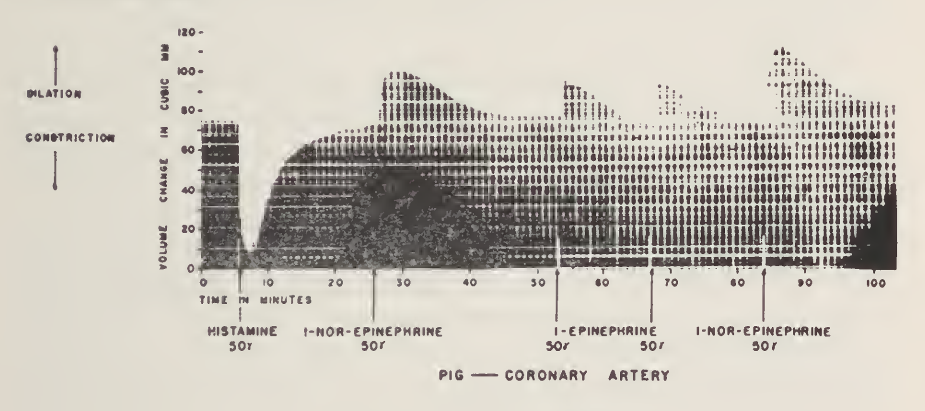


FIGURE 1

Histamine first produced vasoconstriction. Vasodilation was then caused by l-norepinephrine, l-epinephrine, l-epinephrine again and finally by l-norepinephrine. Note the reproducibility of responses (Smith, Syverton, & Coxe[27]).

D.—**Levophed does not Increase Cardiac Output or Oxygen Demand.** Either one of these effects, if present, would be undesirable since they would increase the need of the heart for oxygen. It has been clearly demonstrated by cardiac catheterization in man that an infusion of levo-arterenol does not increase and may even decrease cardiac output.[28] In other words, the essential action of nor-epinephrine is to reroute the blood to more vital areas rather than just increasing blood flow generally throughout the whole body. In some experimental animals under highly unnatural conditions, vagotomy, etc., nor-epinephrine has been reported to increase cardiac output, but this has no bearing on its ordinary clinical use.

In regard to general metabolic effects, it is clear that epinephrine increases the basal metabolism in man and that Levophed does not.[30-32] This again represents an advantage for Levophed since any increase in metabolism requires an increased cardiac out-

put and more work by the heart in order to supply the extra oxygen needed for the tissues.

E.—**Levophed has a Positive Inotropic Effect.**—The fact that Levophed is capable of increasing the force of cardiac contraction may be of very considerable importance if any degree of cardiac failure is present. This positive inotropic effect would serve to supplement the action of digitalis. The positive inotropic effects of Levophed has been demonstrated in intact dogs following intravenous administration[33] and on the isolated papillary muscle of the cat.[34]

F.—**Levophed has Proved Effective in Experimental Coronary Occlusion in the Normotensive Dog.**[35] The oxygen tension in the myocardium was measured polarographically in the exposed left ventricles of anesthetized dogs. "Coronary occlusion or severe narrowing is followed by a progressive fall in local oxygen tension, failure of muscle contraction and later characteristic changes in the epicardial electrocardiogram. Intravenous injection of 0.5 microgram per kilogram of l-norepinephrine reverses these evidences of ischemia, while producing marked bradycardia and a slight increase of mean blood pressure but no ventricular ectopic beats. "L-norepinephrine produces greater and more rapid favorable changes in ischemic areas than pure oxygen inhalation. However, oxygen increases the improvement when administered simultaneously. Both agents in combination may produce rises of oxygen tension in large ischemic areas which have failed to show responses to either agent alone."[35] *

G.—**Experimental Myocardial Infarction with Shock is Markedly Improved by Relief of Shock.** In dogs after ligation of the coronary artery the development of shock caused the amount of blood in the ischemic area to be markedly reduced. Moreover, as the shock developed there was marked ballooning of the myocardium which was relieved when the blood pressure was raised by blood transfusions.[36] Shock **per se** of any duration also acts as a myocardial depressant by decreasing the oxygenation of the heart wall.[37] Although the heart may not be in failure, it will require a greater oxygen consumption to do the same amount of work. Hence, it is most important that shock be relieved in the patient suffering from infarction. Because the oxygen tension in the in-

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* In a manuscript received after this paper was written, Frank et al[63] report that in dogs in hemorrhagic shock Levophed Infusion increased the coronary sinus blood flow from 43cc. per min. during shock, to 170 cc. per min. This latter figure was 2½ times the normal rate of flow before shock. The oxygen saturation in the coronary sinus blood was increased at the same time from 14% to 32%.

farcted areas is increased by Levophed infusions, it is very probable that the size of the infarct is reduced, particularly at the periphery of the lesion, where increases in the oxygen supply may allow the anoxic tissue to recover.

CLINICAL RESULTS ON THE TREATMENT WITH LEVOPHED OF MYOCARDIAL INFARCTION WITH SHOCK

A summary of data on 115 patients treated with Levophed by 14 investigators is presented in Table 2.[38-51] Some of the data are not completely comparable. However, insofar as the information reported permitted, the data from each author were broken down according to the severity of the shock and the nature of the complications. In the case of 39 patients, there was insufficient data for accurate classification.

The last two columns in Table 2 compare survival rates with and without Levophed. The values for the cases without Levophed were obtained by summarizing survival rates under various therapeutic regimens[52-57] reported by a number of cardiologists. There is a wide variation in the reported survival rates but it would appear that most of this variation was due to differences in definition. Master et al.[53] report a 50% survival rate in myocardial infarction with shock as do Levine and Brown.[57] On the other hand, Hellerstein[52] reports only a 12% survival. Master et al. and Levine and Brown consider anyone with pallor and/or cyanosis, tachycardia, a weak pulse and a cold moist skin as being in shock. Hellerstein et al. require that, in addition to the usual clinical signs and symptoms, the systolic blood pressure must be below 80 mm. Hg. for more than one hour. A low blood pressure without other clinical signs would not be classified as shock. Using the criteria of a blood pressure under 80 mm. Hg. and clinical signs and symptoms of shock, a review of the case reports of Levine and Brown shows that they had 10 cases out of 145 who fitted this criteria, all of whom died. A later publication of Levine's confirms this, since a survival rate of 7% was reported for patients with severe shock.[56]

Epstein and Relman[54] using criteria of signs and symptoms plus blood pressure below 90 mm. Hg. had a survival rate of 10% independent of the presence of failure. Mintz and Katz[55] did not specify their criteria but reported a 22% survival in patients with shock. The report of Beckwith[41] indicates that ventricular arrhythmia carries a poorer prognosis than no arrhythmia but he does not report on arrhythmia plus shock.

A comparison of the data on survival rates without Levophed

TABLE 2

A SUMMARY OF CLINICAL DATA*

LEVOPHED ADMINISTERED BY CONTINUOUS INTRAVENOUS DRIP

4 mg. (one ampul) per liter of 5% dextrose solution. Average maintenance rate 30 drops per minute.

Patient's Condition	Number of Patients	Average Systolic Blood Pressure mm.Hg.	Levophed Response			Duration of Infusion			Survival			Survival Rate Without Levophed %	Survival Rate With Levophed %
			Good	Poor or Absent	Un-known	0-1 Hour	Over 1 Hour	Average Hours	Lived	Died	Un-known		
Infarction Plus:													
Mild Shock (B.P.80-40)	19	68	19	0	0	1	17	31	10	7	2	15	53
Severe Shock (B.P.40-0)	32	0	22	8	2	6	26	29	10	21	1	5	32
Shock & Failure	14	58	11	3	0	2	6	31	--	--	14	10	
Shock & Arrhythmia													
auricular	9	18	9	0	0	0	9	27	5	3	1	20	55
ventricular	2	35	2	0	0	0	2	24	1	0	1	20	50
Shock, But Conditions Not Definitely Known	39	--	14	3	22	--	--	23	14	12	13	25 to 50	36
TOTALS	115		77 (67%)	14 (12%)	24 (21%)	9	60	28	40	43	21	20	39

* Tabulated from the data of:

38. Miller & Baker
39. Kurland & Malach
40. Skelton, Mills & Moyer
41. Beekwith
42. Moyer, Skelton & Mills
43. Livesay & Chapman
44. Cochran, Wallace & Griffith
45. Sampson & Zipser
46. English
47. Miller, Shifrin, Kaplan Gold, Billings & Katz
48. Schoolman, Pascale, Bernstein & Littman
49. Calenda, Uricchio, & Friedman
50. Smith & Guz
51. Gazes, Goldberg & Darby

with our present data, where Levophed was used, clearly reveals that Levophed is effective, and indeed life saving in many cases. In fact, the summary shows that Levophed restored the level of blood pressure in 67% of the patients treated. If from this comparison those cases are omitted where the blood pressure response was not reported, we have 77 out of 91 (85%) patients who had good circulatory response to the infusion of the drug.

Equally dramatic is the effect on survival from the acute attack. In mild shock cases, where only 15% of patients ordinarily survive, 53% survived when Levophed was used. Levophed here more than tripled the chance of recovery from this degree of myocardial shock. In the severe shock patients, most of whom were apparently pulseless, with no perceptable systolic pressure, only 5% ordinarily survive with conventional therapy. In this particularly hopeless group Levophed produced good blood pressure levels in 22 out of 30 patients and resulted in 32% of the patients surviving their acute episode. This new circulatory hormone in the latter cases gave the patient a six fold better chance of surviving the immediate effects of his coronary occlusion.*

These important therapeutic results tremendously improve the immediate prognosis of the coronary patient. They indicate that much is to be gained by vigorously treating his shock and anoxemia with every available resource.

CAUTIONS TO BE OBSERVED WITH LEVOPHED

1. **Congestive Failure.** This should be corrected with digitalis while giving Levophed. Levophed itself has a positive inotropic effect; but, inasmuch as it is apt to increase the work of the heart, the use of digitalis would be advisable in the presence of failure.

2. **Hypertension persisting Despite the Infarction Contraindicates Levophed.** However, Levophed may be valuable if there has been a considerable drop in pressure even though the patient is not in shock.

3. **Blood Pressure and Infusion Rate must be Checked Regularly.** Once the proper infusion rates has been achieved, the patient should be checked every 15 minutes. Levophed has a very

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* Since this paper was prepared Binder et al.[62] have reported an additional 25 cases of severe myocardial shock. In their hands, also, 32% of the patients treated with Levophed recovered from the acute severe shock. With the best alternative treatment they saved only 6 out of 57 patients, or a survival rate of 10.5%. Therefore, in their hands Levophed trebled the chance of survival.

fast onset of action, 30-60 seconds, and a very short duration, 3-4 minutes. If the infusion is too fast, hypertension with ectopic beats or congestive failure may occur; and, if too slow, the patient may relapse into shock.

4. **Sloughs May Occur if Levophed Leaks Out Into Subcutaneous Tissues.**—Leg veins should not be the first choice for Levophed infusions. Leakage of Levophed into the tissues either around the needle or through the vein wall, may cause a slough. Experience has indicated that this is most apt to occur in the presence of peripheral vascular disease and particularly when leg veins are used. Recently it has been reported that the risk of sloughing is diminished if the Levophed is introduced through intravenous polyethylene tubing which is inserted for some distance into the vein.

If subcutaneous leakage does occur, the skin overlaying the area will be markedly blanched. To prevent a slough the infusion should be shifted to another site and the area of blanching infiltrated with 2% procaine, starting from the periphery of the blanched area. The area should probably be neither warmed nor chilled. Chilling tends to increase vasoconstriction although it decreases metabolism. Warming does the reverse. In any one situation it is hard to predict which effect would be most prominent.

5. **Theoretically Levophed may Increase the Danger of Ventricular Rupture by Increasing Pressure.** Rupture is most likely to occur 5-8 days after infarction,[58] whereas Levophed is usually needed early and may improve oxygen supply sufficiently to prevent the necrosis predisposing to rupture.

Some people have objected to the use of Levophed because of the hypothetical risk of inducing ectopic beats and arrhythmias. In the cat, under hydrocarbon anesthesia, epinephrine and Levophed are equally effective at inducing ventricular fibrillation.[59] However, under these conditions anything which produces hypertension may induce fibrillation, and it is not a specific effect of Levophed. In the dog with the coronary artery ligated, the induction of too great a rise in blood pressure may cause ectopic beats.[35] However, an infusion causing only a gradual rise in pressure will produce a beneficial effect. In man Levophed has proved effective in controlling ventricular and auricular tachycardia[48] presumably by improving oxygenation and eliminating anoxic foci. Moreover, in patients with complete heart block Levophed has very little effect on the heart rate whereas epinephrine has a marked effect.[60] This is in contrast to the dog where Levophed and epinephrine exert equal positive chronotropic effects. By improving coronary

circulation Levophed has proved effective in preventing ectopic beats due to anoxia. However, a large excess of the drug which produces severe hypertension may of itself induce ectopic beats.

6. **Levophed Should be Diluted with Glucose Solution Rather than with Saline for Infusion.** Experience has shown that in saline solutions, where air may be bubbling through, as in infusion bottles, the Levophed gradually oxidizes. This results in the need to administer continuously increasing volumes of solution to maintain an even level of pressure. When glucose solution is used to dilute the Levophed or glucose in saline is used, such oxidation is largerly prevented and more stable levels of inflow are therefore possible. The concentration of the Levophed infusion solution may be increased as required if the patient needs so much of the compound that the excessive fluid volume becomes a problem.

PROPERTIES OF OTHER VASOCONSTRICTORS WHICH PRECLUDE THEIR USE IN MYOCARDIAL INFARCTION

1. Increase Cardiac Output.
2. Increase Oxygen Consumption.
3. Increase Cardiac Irritability.
4. Increase Heart Rate.
5. Cause Coronary Constriction.

In addition, other vasoconstrictors have been tried and largely discarded in the treatment of myocardial infarction because of tachyphylaxis (a decreasing effectiveness on repeated administration) and ineffectiveness. In regard to the latter point there are no cases reported where Levophed has failed to raise the blood pressure and other pressor amines succeeded, whereas there are numerous examples of the reverse, i.e., where Levophed succeeded after the others had failed.

LEVOPHED IN SURGICAL AND OTHER SHOCK

The use of Levophed in shock from myocardial infarction developed after its wide and extensive use in surgical and other shock. Its effectiveness and safety in these have been clearly established. An example of its use in shock due to perforated peptic ulcer is shown in Fig. 2.

Shock may be classified into two main groups, blood or plasma loss and vasodilator shock. A third somewhat controversial category has also been proposed — shock due to cardiac failure, so

Fremont, Luger, Surks & Kleinman

Fig. 2

called "forward failure." A summary of the use of Levophed in these categories of shock is outlined below.

A. **Blood or Plasma Loss Shock** is best treated by replacing blood and plasma. Lacking these, or until available, Levophed will maintain pressure and circulation to kidneys, brain and heart by constricting the venous reservoir, limiting blood flow through splanchnic and skeletal muscle blood vessels and by pulling fluid out of the tissues. Moreover, its vasoconstrictor action may limit plasma and fluid loss into the tissues.

B. **Vasodilation Shock** due to decreased vasomotor tone, reflex vasodilation, drugs, toxins, allergic reactions and adrenal failure is best treated by constricting the vascular system rather than filling it with blood and plasma. In the event of adrenal failure, adrenal cortical extract or cortisone should be given with Levophed since it potentiates the action of Levophed.

C. **Cardiogenic Shock** due to inability of the heart to maintain cardiac output is best treated by limiting the available output to the most vital areas, i.e., Levophed to maintain blood pressure, and hence coronary flow, and also renal and cerebral flow.

SUMMARY AND CONCLUSIONS

1) Intravenous infusion of levo-arterenol (Levophed) greatly increases the chances of survival in patients who have gone into

shock following myocardial infarction from coronary spasm or occlusion.

2) In mild cases of myocardial shock where the expected survival rate is 15%, Levophed infusions more than triple this to 53%.

3) In severe pulseless cases of myocardial shock where the usual survival rate is only 1 out of 20 cases, Levophed infusions permits recovery from the acute episode of one out of every three cases.

4) Similar degrees of benefit in other varieties of shock have been reported from Levophed administration.

5) Therefore, the use of Levophed in all the frequent varieties of circulatory shock has been found to contribute dramatically to recovery of a large proportion of the patients and to markedly reduce mortality from this condition.

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ANAPHYLACTOID SHOCK DUE TO PENICILLIN

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Since the advent of the antibiotics, the incidence of allergic reactions to these agents has increased steadily. Physicians using these drugs have encountered many instances of urticaria, angio-edema and asthma arising secondary to their use. These episodes usually respond favorably to therapy, and as a rule do not end-anger the life of the patient. There is another less frequent, but very severe type of allergic reaction, which carries with it an appreciable mortality. This is anaphylactoid shock, so termed, because of the great similarity of its signs and symptoms to the anaphylactic shock produced experimentally in sensitized laboratory animals, specially the guinea-pig.[1, 2]

In recent years there have appeared in the literature reports of both fatal and non-fatal reactions of this sort, following the use of Penicillin.[3, 7, 10, 12] Anaphylactoid Shock secondary to penicillin is thus becoming more frequent with the indiscriminate use of this antibiotic at times self-prescribed by patients for minor-ailments. Although the majority of the cases mentioned in the literature are secondary to injected penicillin, it must be borne in mind that anaphylactoid shock has occurred following its use topically[10, 12] orally[9] and aerosol.[11]

The signs and symptoms of anaphylactoid shock in humans have been described recently by Siegal et al,[5] Curphey,[4] Feinberg et al,[6] and others, following Waldblott's[3] first report in the literature in 1949. Welch and his co-workers[9] from the U.S. Food and Drug Administration, collected a series of 63 cases in a study which covered 95 American Hospitals over a period of two years. These authors report that as a rule, the patients develop the signs and symptoms shortly after the administration of the offending substance, i.e., penicillin. The most common manifestations are: tightness or a sense of constriction in the chest, dyspnea, intense itching or burning sensation in the skin and or in the mouth and throat. These symptoms are associated with cyanosis and evidences of circulatory collapse usually of severe degree. Instances of frank pulmonary edema have also been encountered by these authors. Urticaria, angioedema, and acute edema of the larynx may also occur as part of the reaction. Kern and Wimberly,[8] Feinberg et al,[6] and others, have described signs and symptoms attributable to

CNS involvement, manifested by stupor, deliriousness, coma, and convulsions.

The above manifestations face us with desperately ill patient, making mandatory to institute urgently an adequate treatment in order to prevent a fatal outcome.

A case of anaphylactoid shock secondary to penicillin administration occurred recently at the Hospital Dr. M. Figueroa at Arecibo, P. R. Since this type of reaction will probably be seen with increasing frequency in the future, it is our purpose to bring it to the attention of the Medical profession of the Island.

CASE REPORT

A 44 year-old hospital employee received a self-prescribed, long-acting procaine penicillin injection of 1,200,000 units. Within two minutes, the patient felt nauseated, very weak and fell down. His complaint at the time was tightness in the chest. He rapidly became very cyanotic and began to gasp for air; was taken to the O. R. with pulse and blood pressure unobtainable, unconsciousness, swollen tongue, and a labored, stertorous breathing associated with a pink, frothy sputum at the mouth and nostrils.

Auscultation of the chest disclosed fine and coarse rales; the heart sounds were distant and weak. Generalized clonic and tonic convulsions developed shortly after. He immediately received epinephrine and coramine intramuscularly; in addition he was administered oxygen by nasal catheter, and aspiration of nasopharyngeal airways was done. Soon thereafter he was given ouabain intravenously, Chlor-trimeton maleate intramuscularly, and 5% Glucose in water intravenously to combat the circulatory collapse. About 10 minutes after institution of the therapy, the condition of the patient began to improve slowly. The cyanosis slowly disappeared, he recovered consciousness and began to talk somewhat incoherently, though his pulse and blood pressure were still unobtainable. Sixty minutes after the onset of the reaction a thready radial pulse could be felt and BP was 60/0. From this point on therapy consisted of intravenous administration of 5% G/W, epinephrine, and antihistaminics at regular intervals.

Five hours after the onset of his signs and symptoms the patient was much improved, acyanotic, breathing comfortably, with a steady rise in blood pressure and with all evidence of pulmonary edema absent. At this time he voided about 175 cc. of clear urine. Laboratory studies including CBC, urinalysis, NPN, EKG, and chest X-ray performed at that time and in the next day a erythrocyte count of cu. mm. were all within normal limits, except for a Hgb. at 134% and 6.5 million.

Subsequent laboratory examinations two days later were all within normal limits.

After the initial critical period the patient had a rapid and favorable recovery and was discharged well two days later.

COMMENT

Anaphylactoid shock, a very severe and at times fatal type of allergic reaction, is nowadays being described with alarming frequency secondary to the use and misuse of penicillin. It can follow systemic as well as local use of the antibiotics. Clinically, the reaction maybe manifested by the presence of shock, cyanosis, dyspnea, pulmonary edema, urticaria, angiodema, stupor or convulsions. These patients are critically ill and the condition carries with it an appreciable mortality rate. The treatment of anaphylactoid shock varies, but in general there is agreement as to the use of epinephrine, oxygen, antihistaminic drugs (parenterally if necessary), adequate I.V. fluids and other general measures such as warmth, posture, aspiration of secretions as the case may be.

One has only to realize the inherent menace to the patient's life in anaphylactoid shock to be convinced that the careless use of penicillin in patients with known sensitivity to it, is extremely dangerous. The physician who carefully investigates the possibility of an allergic response on the part of the person to whom he plans to prescribe penicillin will sometime be rewarded by not bringing upon his patient the threat of fatal or near-fatal anaphylactoid shock.

SUMMARY

A case of acute anaphylactoid shock due to penicillin is reported. The signs and symptoms of this severe allergic reaction are described, together with the treatment. Caution should be exercised in administering penicillin to patients with known sensitivity to this antibiotics as a means of preventing anaphylactoid shock.

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CONFERENCIA CLINICOPATOLOGICA

PRESENTACION DEL CASO

Primera Admisión:

S. J. C. H. - 104147

R. S., un jovencito blanco de 17 años de edad fué admitido al servicio de neurocirugía en septiembre de 1952 quejándose de dolores de cabeza, diplopia, vértigo y anorexia.

El enfermo había sido atendido anteriormente en el Hospital Municipal de Río Piedras debido a "anemia de severidad moderada", pero todavía no había recibido medicación alguna. Durante el último año había sufrido de cefaleas intensas generalizadas y frecuentes, aunque a intervalos irregulares, las cuales se hicieron más severas y casi contínuas, con localización en el lado derecho, durante los últimos tres meses. Alrededor de un mes antes notó por primera vez diplopia, vértigo y anorexia.

El examen físico demostró los siguientes datos: T-99ºF; P-80; R-20; PA-105/70. Había papiledema bilateral, debilidad de ambos rectos laterales con un pequeño estrabismo y una reacción pupilar débil a la luz y a la acomodación. Los reflejos estaban uniformemente disminuídos. El resto del examen fué negativo.

Los exámenes de laboratorio demostraban: hematíes - 3.36 M.; hemoglobina - 54%; leucocitos - 7,350 con un diferencial normal; orina - 4-8 hematíes y 15-20 leucocitos por campo y muchas bacterias en el sedimento; Khan - negativo en dos ocasiones; heces - T. Trichiura; Necator Americanus y Ascaris lumbricoides. Tiempo de protrombina 19 seg. (control 15 seg.). El líquido cefalorraquídeo, cuya presión inicial estaba aumentada, demostró unas proteínas totales de 19 mg. y glucosa 79 mgs. %. No había células blancas pero sí muchos hematíes.

Un arteriograma percutáneo fué negativo y la ventriculografía demostró solamente que los ventrículos laterales estaban un poco más pequeños de lo normal.

Durante esta admisión el enfermo recibió dos transfusiones de sangre para un total de 750 cc. Un segundo hemograma demostró 3.69 M de hematíes con 75% de hemoglobina y 2,350 leucocitos con un diferencial normal. Unos días más tarde se repitió este último recuento y dió 7,250 con 16% eosinófilos y 38% linfocitos. Fué dado de alta mejorado un mes después de admitido.

Segunda Admisión (4 meses después)

El enfermo continuó prácticamente bien hasta dos semanas antes en que desarrolló un catarro con tos y expectoración de es-

puto amarillento. Ocho días más tarde notó la orina de color oscuro y al día siguiente notó amarillez de las escleróticas. Por lo demás se encontraba bien.

El examen físico reveló T - 98.6ºF, P - 80; R - 20, PA - 122/60. Ligera coloración ictérica de la piel y los ojos. El hígado se palpaba a 3.5 cm. por debajo del reborde costal derecho en la línea clavicular media y no estaba doloroso a la palpación. El bazo se palpaba ligeramente en la inspiración profunda.

Los exámenes de laboratorio revelaron los siguientes datos: hematíes - 3.31 M; hemoglobina - 60% ; leucocitos - 3,600 con un diferencial normal. Orina - albúmina - 2+, glucosa - negativa; 1-2 hematíes, 6-8 leucocitos y muchas bacterias por campo. Urobilinógeno pos. 1:10 y trazas hasta 1:40. Bilirubina - 0.48 mg. en 1 min. y 1.68 mg. en 30 min. Kahn - negativo. Turbidez del timol - 4 unidades. Preparación para deformación falciforme (sickle-cell prep.) - negativa. La orina posteriormente aclaró por completo.

El tratamiento consistió en reposo en cama, dieta alta en proteínas e hidratos de carbono, y baja en vitaminas y extracto hepático grasa. A los diez días fué dado de alta muy mejorado y con instrucciones de continuar el régimen en su casa y asistir regularmente a la clínica.

Tercera admisión: (un año después).

El enfermo continuó bajo observación durante este intervalo de tiempo aunque no siguió los consejos e indicaciones terapéuticas, habiendo trabajado contínuamente. Varias determinaciones de bilirubina durante este tiempo oscilaron entre 0.36 y 0.48 mg.% en un minuto y 1.68 y 3.12 mg.% en 30 minutos. La floculación de cefalina variaba entre 3 y 4+. Un mes antes de esta admisión empezó a notar nuevamente color oscuro de la orina e ictericia de las escleróticas además de cansancio, debilidad general, anorexia y pérdida de peso. También había notado un dolor sordo, intermitente y moderadamente severo en el cuadrante superior izquierdo.

El examen físico reveló una marcada desnutrición y moderada ictericia. El hígado se notaba reducido de tamaño a la percusión según un observador y aumentado de tamaño según otro (ambos exámenes en distintas fechas). El bazo siempre se encontró de 7 a 8 cm. por debajo del reborde costal izquierdo. El abdomen estaba un poco distentido y se notaba más pequeña cantidad de líquido libre en la cavidad peritoneal. Los orificios de las trepanaciones se hallaban distendidos.

Los exámenes de laboratorio: hematíes - 3.05 M; hemoglobina - 55% ; leucocitos - 1,750 (PMN - 48; L - 36; Eos - 16). Orina -

esencialmente negativa excepto por trazas de bilis. Proteínas totales del suero 7.7 gm. (Alb - 4.5; Glob. - 3.2). Fosfatasa alcalina - 2.73 unidades Bodansky. Heces - ascaris y estrongiloides. Tiempo de protrombina 16.6 seg. (control - 14 seg.). Kahn - neg. Varias determinaciones de plaquetas oscilaron entre 66,000 y 96,000; las pruebas de Hanger entre 3 y 4 + y la bilirubina entre 0.24 y 0.6 mg.% en 1 minuto y 0.48 y 3.1 mg.% en 30 minutos. Los tiempos de sangría y de coagulación estaban dentro de lo normal. Varios exámenes de excreta fueron negativos para S. Mansoni.

Durante esta admisión recibió el mismo tratamiento de reposo, dieta alta en hidratos de carbono y proteínas, suplementos de vitaminas y extracto hepático. Fué dado de alta a los dos meses a pesar de que había habido muy poca mejoría.

Cuarta Admisión: (3 meses después).

El enfermo continuó en las mismas condiciones y en la imposibilidad de continuar el tratamiento en su casa, fué readmitido con los mismos síntomas que la vez anterior.

El examen físico reveló esencialmente los mismos hallazgos que en la tercera admisión excepto que ahora el hígado estaba definitivamente pequeño y el bazo sólo alcanzaba unos 5-6 cm. bajo el reborde costal.

Las pruebas de laboratorio también fueron esencialmente igual que en la tercera admisión aunque las plaquetas llegaron a bajar a 24,000 y los leucocitos a 1,500. El colesterol fué 171 mg.% y la prueba del timol 3.2 y 4.3 unidades en dos ocasiones.

Un estudio radiográfico del esófago demostró várices esofágicas y una radiografía del abdómen confirmó la presencia de una masa en el cuadrante superior izquierdo sin calcificaciones que se intepretó como bazo grandemente aumentado de tamaño.

Esta vez el enfermo recibió varias transfusiones de sangre además del tratamiento indicado anteriormente. Fué dado de alta unos dos meses más tarde ligeramente mejorado.

Quinta Admisión: (un mes después):

Después de salir del hospital el enfermo estuvo relativamente bien hasta el día antes de admitirse en que empezó a notar dolor en ambos cuadrantes superiores acompañado de fiebre y escalofríos pero sin náuseas ni vómitos. Durante el mes anterior había tenido heces negras varias veces.

El examen, además de los hallazgos de las últimas dos admisiones, demostró hepatomegalia definitiva. El bazo llegaba a unos

5-6 cm. debajo del borde costal y estaba ligeramente doloroso a la palpación.

Las pruebas de laboratorio fueron más o menos como en las dos últimas admisiones pero además se notó una reticulocitosis variando entre 3 y 5%. El examen de la médula ósea reveló una reacción hiperplástica regenerativa. Una determinación de colesterol fué de 433 mg.%.

Se le dió el mismo tratamiento que en su cuarta admisión y se consultó al departamento de cirugía sobre la posibilidad de practicarle una esplenectomía y anastomosis porto-cava pero los cirujanos rehusaron intervenir debido a la mala condición general del enfermo.

Mejoró grandemente después de las transfusiones y hasta llegó a trasladarse al departamento de cirugía con vistas a operarse pero continuó teniendo febrícula por lo cual fué de nuevo rechazado y devuelto al departamento de medicina. Decidió marcharse del hospital por su cuenta y en contra de los consejos médicos, unos cinco meses después de haber ingresado.

Sexta y última admisión: (5 días después).

Regresó el enfermo en muy malas condiciones quejándose de fiebre, dispnea y distensión abdominal. Los hallazgos físicos fueron iguales a las últimas admisiones excepto que la malnutrición había progresado al igual que la cantidad de líquido ascítico. La piel mostraba una red venosa superficial bien manifiesta.

Los exámenes de laboratorio revelaron de nuevo anemia, leucopenia y trombocitopenia; ligera hiperbilirubinemia y sangre oculta en la excreta.

El enfermo empeoró rápidamente desarrollando una diarrea sanguinolenta y luego se tornó inconsciente. Resistía, sin embargo, los esfuerzos de los médicos para pasarle el tubo naso-gástrico para alimentarle y dos veces se lo sacó. Finalmente hubo que amarrarlo. Se le dieron transfusiones, infusiones, etc., a pesar de lo cual continuó deteriorando hasta que por fin murió al trigésimo día de hospitalización y dos años y medio después de la primera admisión.

DISCUSION CLINICA

Dr. Manuel Paniagua:

La primera lectura de este protocolo nos da la impresión de que se trata de un caso bastante sencillo.

En primer lugar tenemos una admisión al servicio de neurocirugía con un cuadro de cefalalgia, diplopia, vértigo y papiledema. Estudios minuciosos incluyendo un arteriograma y un ventriculograma son negativos y el líquido cefalorraquídeo solamente demuestra un aumento de presión. Este cuadro clínico encaja muy bien con el llamado síndrome de Quincke o hidropesía hipertensa de las meninges. Es ésta una condición de etiología desconocida aunque en algunos casos se ha encontrado una obstrucción del seno sagital que entorpece el desagüe normal del líquido cefalorraquídeo, causando el cuadro clínico descrito anteriormente. Durante esta hospitalización se encuentra una anemia moderada para la cual recibe dos transfusiones de 500 y 250 ml. respectivamente.

Dado de alta, continúa en aparente estado de salud hasta unos cuatro meses más tarde cuando, una o dos semanas después de una afección respiratoria, desarrolla lo que se interpreta como hematuria acompañada de ictericia. En esta ocasión el hígado se encuentra aumentado de tamaño y el bazo se hace palpable. El cuadro evoluciona como una hepatitis infecciosa, probablemente relacionada con las transfusiones de sangre recibidas durante su primera admisión, y el enfermo recibe el tratamiento indicado para esta enfermedad.

De nuevo es dado de alta pero no sigue las instrucciones médicas y regresa un año después con un cuadro sugestivo de cirrosis. Subsiguientemente desarrolla esplenomegalia, pancitopenia, várices esofágicas con repetidas hemorragias, ascitis y después de otros dos períodos de hospitalización muere en aparente estado de insuficiencia hepática unos dos años y medio después de la primera admisión.

El desarrollo de este caso es aparentemente obvio; tan obvio que inmediatamente me hace dudar seriamente este diagnóstico. He participado en bastantes de estos ejercicios clínico-patológicos para sospechar de lo obvio. Por otra parte, cuando repasamos el protocolo con un poco de cuidado, encontramos algunos datos que no concuerdan con este diagnóstico.

Durante la segunda hospitalización con el cuadro de aparente hepatitis, encontramos solamente una prueba de Hanger y una de turbidez del timol, ambas con resultados negativos o normales. Estos resultados están en franca discrepancia con la impresión clínica y aunque soy de los que creo que ésta última es más importante, no dejo de tener en cuenta esta aparente paradoja en la evaluación total del caso.

Hay otro detalle de laboratorio que me hace dudar grandemente al diagnóstico de cirrosis; a través de toda su evolución "cuesta abajo", este enfermo mantuvo un nivel de albúmina en el

suero por encima de 3.5 gm. y una proporción de albúmina a globulina de más de uno. Esto, a mi parecer, es casi incompatible con el diagnóstico de cirrosis. Tampoco va con cirrosis una retención de BSP de solo 6%.

Por otro lado, el episodio final que precedió a la muerte estuvo caracterizado por intranquilidad, irracionalidad y sobresalto más sugestivos de hemorragias que de coma hepático.

Si volvemos de nuevo sobre algunos detalles del protocolo veremos que este enfermo ha padecido de anemia desde antes de la primera hospitalización. Esta anemia, de tipo hipocrómico, se mantiene en un nivel más o menos estable (entre 2 y 3.5 millones de hematíes y entre 5 y 9 gms. de hemoglobina) a través de toda la enfemedad. Una anemia de este tipo en un individuo bien nutrido, como se describe el enfermo en su primera admisión, merece una investigación más completa antes de proceder a transfusiones. De ahí en adelante se hace más difícil establecer un diagnóstico exacto del tipo de anemia; sin embargo, el hecho de que está acompañada de ictericia y de esplenomegalia me hace pensar en la posibilidad de una anemia hemolítica. La ictericia de variable intensidad y la anemia que se acentúa con su aparición podrían ser debidas a pequeñas crisis hemolíticas. La leucopenia y trombopenia pueden ser explicadas por el hiperesplenismo. La médula ósea hiperplástica también es compatible con este diagnóstico como es el hecho de que mejorara con las transfusiones.

Solamente mencionaré dos de las anemias hemolíticas por parecerme las más posibles en este caso. En primer lugar la anemia hemolítica congénita; la edad del enfermo y la prueba de Cooms negativa están ambas en favor de esta posibilidad. Por otro lado, la anemia depranocítica quizás podría explicarnos mejor algunos de los fenómenos trombóticos que, como diré más adelante, creo que son muy patentes en este caso. De hecho, esta atractiva hipótesis podría explicarnos el cuadro de hipertensión meníngea de la primera admisión, debido a una posible trombosis del seno venoso sagital. Es cierto que la raza del enfermo y una prueba negativa de deformación de los hematíes están en contra de este diagnóstico pero eso no lo elimina por completo. La prueba puede ser negativa en casos de anemia depranocítica ¿no es así, Dra. Ferrer-Piñero?

Dra. Lillian Ferrer-Piñero — Así es.

Dr. Paniagua — Y esto nos trae a una tercera posibilidad que voy a mencionar sólo para descartarla; la púrpura trombótica trombocitopénica, caracterizada por anemia, esplenomegalia, púrpura y fenómenos trombóticos especialmente en el sistema nervioso central. La duración de la enfermedad y la ausencia de púrpura están en contra de este diagnóstico.

Y llegamos ahora al último episodio. La descripción de la etapa final me parece un poco violenta para un coma hepático. La intranquilidad, sobresalto y desorientación del enfermo me sugieren mejor una hemorragia —probablemente por várices esofágicas. Sin embargo, los dolores sordos en el abdomen superior, el rápido aumento de la ascitis y las diarreas con sangre me hacen pensar en una posibilidad muy atractiva: una trombosis gradual de la vena porta. Esto explicaría todo el cuadro clínico incluso la hipertensión portal, esplenomegalia, pancitopenia, várices esofágicas y hemorragia final.

Resumiendo, diré que no creo que este enfermo tuviera cirrosis. Puede que tuviera una hepatitis aguda pero no es probable. Me parece que el cuadro es uno de anemia hemolítica, posiblemente drepanocítica, con fenómenos trombóticos incluyendo un episodio de trombosis del seno venoso sagital, trombosis de la vena porta con hipertensión portal, pancitopenia secundaria e hiperesplenismo y rotura de várices esofágicas con hemorragia como episodio final.

Dr. Darío Del Nero — ¿Ha considerado usted la posibilidad de una trombosis de la vena esplénica; el llamado síndrome de Banti?

Dr. Paniagua — El llamado síndrome de Banti es cuadro clínico caracterizado por esplenomegalia con o sin hepatomegalia, ascitis y várices esofágicas que en la inmensa mayoría de los casos es debido a una cirrosis hepática. Ya dije que no creo que este enfermo tuviera cirrosis. En cuanto a la posibilidad de una trombosis de la vena esplénica solamente la creo remota pues no hay antecedentes de trauma. Además, creo que esto es parte de cuadro de fenómenos trombóticos diversos y me parece más probable que la obstrucción sea en la porta aunque posiblemente con extensión a la esplénica.

Diagnósticos clínicos:

1. Hepatitis a virus (debido a suero homólogo)
2. Cirrosis post-necrótica
3. Hipertensión portal
4. Esplenomegalia congestiva con hiperesplenismo
5. Várices esofágicas con rotura y hemorragia

Diagnósticos del Dr. Manuel Paniagua:

1. Anemia hemolítica (probablemente deprenocítica)
2. Trombosis del seno venoso sagital
3. Trombosis de la vena porta

4. Hipertensión portal
5. Esplenomegalia
6. Várices esofágicas con rotura y hemorragia

Dr. Víctor M. Araán: En la necropsia nos encontramos con un hombre caquéctico y con decoloración ictérica de las escleróticas. Había además, edema moderado de las extremidades inferiores. El abdomen estaba distendido y se percibía fácilmente una oleada ascítica. La cavidad peritoneal contenía 5,000 cc de un líquido transparente y amarillento. El bazo pesaba 1.050 gm. y estaba íntimamente adherido a la cúpula diafragmática izquierda. La cápsula esplénica estaba engrosada irregularmente, variando en espesor de 0.5 a 2.5 cm. Además estaba cubierta por un tejido gris blancuzco, firme y duro que cortaba con resistencia. La pulpa esplénica subyacente a las zonas de engrosamiento capsular, aparecía con un color gris azulado y exudaba una cantidad mínima de material hemorrágico. En otras zonas tenía un color rojo oscuro, firme y mate correspondiendo a zonas de infarto, claramente delimitadas del resto del parénquima. Pequeños nódulos de un color pardo obscuro se veían por toda la superficie del corte, a excepción de las zonas de infarto. Los corpúsculos de Malpigio estaban completamente obliterados. Los vasos del hilio esplénico estaban engrosados y dilatados; en la luz se podían ver trombos en estadios distintos de organización. Las paredes de los vasos estaban engrosadas y cortaban con marcada resistencia. Estaban además envueltas en un manto espeso de tejido conectivo que las hacía resaltar claramente. En conjunto ofrecían un aspecto angiomatoide. Disecando las venas esplénicas hacia la pulpa del órgano, pudimos comprobar que la trombosis se extendía hasta la vecindad de los infartos recientes, mientras que aquellas regiones que mostraban tejido conectivo viejo estaban en la vecindad de estructuras vasculares transformadas en cuerdas sólidas. En general se notaba una resistencia marcada al corte del bazo. Desgraciadamente la disección de la esplénica no se hizo también en la dirección de la porta, así que no pudimos asegurarnos de la posibilidad de una extensión de la trombosis a la porta misma. El hígado pesó 1,260 gramos. La cápsula era normal y el órgano carecía de hallazgos macroscópicos a excepción de una apariencia a "nuez moscada" muy típica de los hígados afectos de congestión crónica. Los demás órganos eran normales macroscópicamente. Quisiera hacer aquí un inciso para felicitar al Dr. Paniagua por su discusión que ha sido llevada muy de acuerdo con los hallazgos anatomopatológicos. Como él muy bien observó, el paciente no tenía hepatitis infecciosa. Si tal evento fué parte de su enfermedad en el pasado, no quedaban hitos que nos in-

dicasen siquiera remotamente tal posibilidad. Si hubo hepatitis, ésta en verdad, poco o nada tuvo que ver con lo que causó los síntomas y eventualmente el exitus del enfermo. No había ni infiltración linfocítica, ni zonas de regeneración hepática, ni aumento del retículo, ni distorsión de la arquitectura normal del lóbulo hepático. Había sí un edema manifiesto del espacio linfático de Diesse y un cierto grado de degeneración parenquimatosa reversible, hechos ambos que han sido probados ser resultado de anoxia o hipoxia agonal, por H. Popper y su escuela. Otro dato de más interés y que tiene relación directa con la causa real de la enfermedad en este caso, fué la presencia en varias ramas de la vena porta y de la hepática de trombos en estadios distintos de organización. (Fig. 1) Esto no era algo difuso y que afectase a la ma-

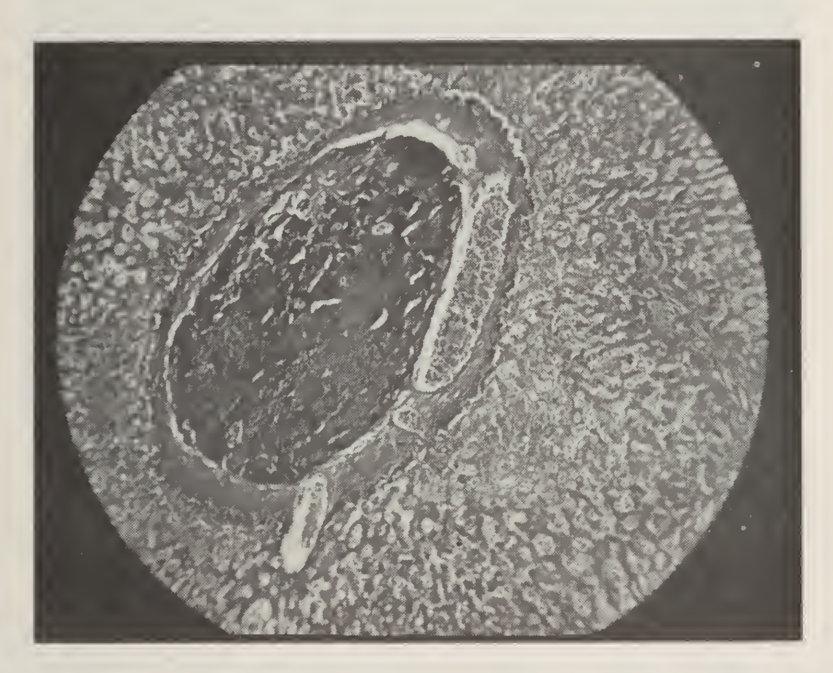


Fig. 1. Hígado. Trombosis de una de las ramas de la vena hepática. Nótese la marcada dilatación de las venas y de sus tributarias, así como la intensa congestión venosa de los sinusoides circundantes. X 360, reticulina de Wilder.

yor parte de las ramas de la porta, sino limitado a unas pocas, como pudimos comprobar en las secciones múltiples que se tomaron. En estas ramas portas se notaba una dilatación marcada de la luz y un ligero aumento del conectivo perivascular, pero sin que este afectase a los otros constituyentes de la triada porta, como se ve en las cirrosis sea cual fuere su etiología. No había pues ni hepatitis, ni cirrosis. El bazo mostraba todos los caracteres his-

topatológicos de lo que se ha venido llamando síndrome de Banti. Además de la desaparición total de los centros germinativos en los folículos se notaba un incremento marcado del conectivo. Esto correspondería a lo que Banti mismo llamó fibroadenia; los senos venosos estaban dilatados, había una hiperplasia de las células reticuloendoteliales y un aumento acentuado de la reticulina como se ve en las impregnaciones argénticas que se hicieron. Los nódulos pardos vistos macroscópicamente, corresponden a lo que se conoce con el nombre de nódulos de Gamna-Gandy (Fig. 2), y que son

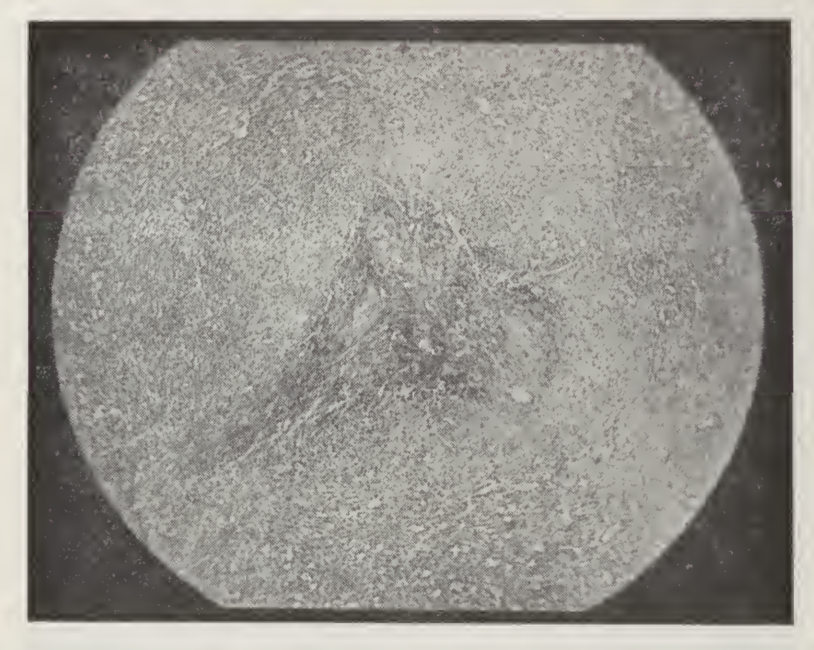


Fig. 2. Bazo. Nódulo de Gamna-Gandy. Obsérvese la fibrosis marcada del estroma esplénico. X 80. Hematoxilina-Eosina.

el resultado final de focos de hemorragia, que se incrustan con calcio y pigmento férrico al mismo tiempo que hay una marcada proliferación de fibras elásticas y del tejido conectivo. Como la precipitación de las sales de hierro y calcio ocurre en las fibras elásticas, éstas aparecen, a primera vista, como estructuras similares a los micelios y ha dado lugar en el pasado a que se atribuyese a una micosis la etilogía del Banti. Tinciones especiales y un estudio más detallado de la histopatología ha demostrado el error de tal aserto. Es preciso recalcar aquí que ninguno de estos hallazgos son característicos de nada. A lo sumo se diría que son típicos de las esplenomegalias congestivas sea cual fuere su origen. Se ven nódulos de Gamna-Gandy en la anemia a células falciformes (sickle cel anemia), en las esplenomegalias congestivas

de tipo cardíaco, en cirrosis con esplenomegalia marcada y en un sin fin de entidades que dan como resultado estasis esplénico y hemorragias secundarias en el parénquima.

Además de estos focos de hemorragias antiguas había muchas zonas de la pulpa con hemorragias más recientes. Pero lo más notable fué el examen de las venas y arterias. Las venas estaban dilatadas y su luz ocupada por trombos (Fig. 3), la mayor

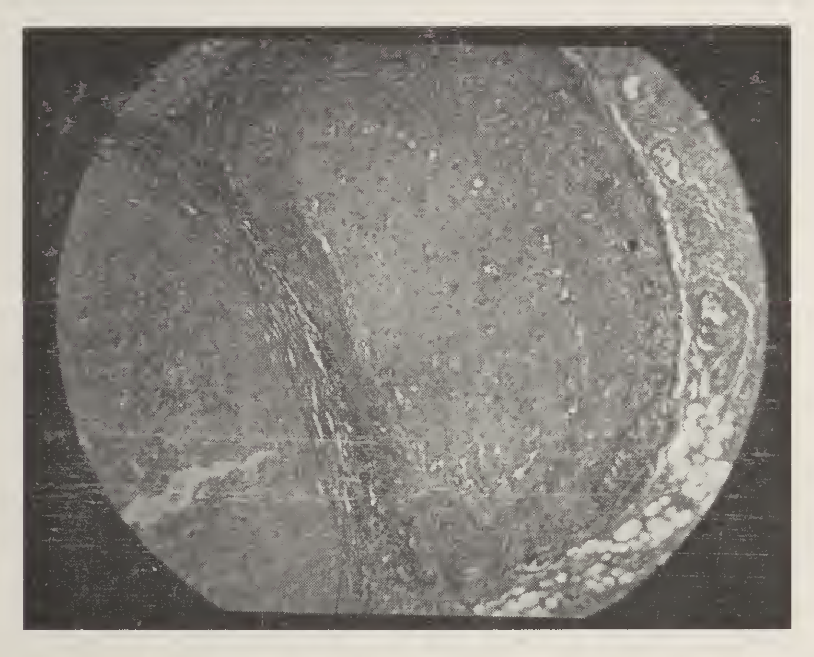


Fig. 3. Bazo. Angiomatosis cavernomatosa y trombosis de las venas esplénicas. La luz de un vaso está reemplazado por tejido conectivo con gran número de capilares. Gran engrosamiento de la pared vascular. X 96, reticulina de Wilder.

parte viejos y organizados e incluso con revascularización incipiente. Otros eran más recientes pero era evidente ya el comienzo de organización. Las arterias eran asiento de un proceso de endarteritis proliferativa y ambas, venas y arterias estaban circundadas por un tejido conectivo abundante y conteniendo un número variable de macrofagos con o sin pigmento férrico ingerido. La apariencia de las venas era la de una transformación angiomatoide típica. Ahora bien, si ésta ocurrió como resultado de la trombosis y si fué la causa de ella es punto difícil de asegurar. ¿Cuál fué pues la causa de la trombosis en este caso? No había historia de trauma, la causa más comun de las trombosis esplénicas; no había historia de enfermedad embolizante que pudiese causar tal trastorno; no había razón para clasificar el caso como anemia a célu-

las falciformes y no existía cirrosis. Osler, hace ya muchos años, puso de relieve la relativa frecuencia con que se ven en la clínica casos de trombosis de la esplénica con cuadro final de insuficiencia hepática y **sin cirrosis.** Hay muchos pacientes que desarrollan este síndrome sin que uno pueda determinar la naturaleza o agente del mismo. Es ésta la razón por la que el nombre de Banti aún permanece en la literatura dando nombre a un síndrome que no siempre puede catalogarse por lo que atañe a sus bases etiológicas. En Egipto, un estudio llevado a cabo por investigadores ingleses puso de relieve la frecuencia de este síndrome en pacientes con esquistosomiasis. En nuestro caso no había evidencia alguna de esquistosoma a pesar de la frecuencia de esta infestación en Puerto Rico. Finalmente nos queda como una razón la posibilidad de que el paciente tuviese una malformación vascular del bazo, lo que se llama transformación cavernomatosa, muy bien estudiada por Moschowitz.

El paciente nuestro tenía a mi juicio desde un principio un síndrome hiperesplénico. Esto explicaría como subrayó el Dr. Paniagua, la anemia y la neutropenia que afectaron al paciente desde un comienzo. Esta anemia quizás explicase también las intensas cefalalgias que sufrió, ya que la anoxemia es factor favorable para cualquier manifestación de tipo cerebral, incluso edema. Y a medida que el bazo aumentó de tamaño y la trombosis se repitió y progresó, así aumentaron los síntomas, primero la acentuación de la anemia, luego los dolores en el hipocondrio izquierdo, más tarde la esplenomegalia y la ictericia que siempre tuvo un carácter hemolítico mas bien que hepatocelular y finalmente la ascitis y las várices esofágicas y la circulación colateral de la pared abdominal. La ruptura de una de estas várices fué el episodio final. Como hallazgo incidental mencionaré la presencia de numerosos trombos en las ramas de la arteria pulmonar, todos ellos recientes y probablemente de origen pélvico o más bien de las venas de las piernas. Esto es algo que ocurre con frecuencia en pacientes caquécticos, gravemente enfermos y como resultado de las trombosis de los plexos venosos.

Naturalmente había también una acentuada nefrosis colémica. El cerebro mostró pequeños focos de encefalomalacia también corrientes en pacientes que han sufrido anoxemia durante un período de varias horas. Por lo demás nada de interés pudo hallarse que pueda explicar directamente la causa de las cefalalgias iniciales.

Otros hallazgos de menos interés y resultado de la enfermedad crónica fueron atrofia testicular y atrofia muscular moderada.

Diagnóstico Anatomopatológico

Trombosis de la vena esplénica y de las ramas de la vena porta y hepática (Síndrome de Banti)
Infartos del bazo, recientes y viejos, múltiples
Esplenomegalia
Periesplenitis
Congestión pasiva del hígado
Embolismo pulmonar difuso
Nefrosis colémica
Varices esofágicas con ruptura y hemorragia gastroentérica
Ascitis
Atrofia testicular
Encefalomalacia, focal
Hiperplasia de la médula ósea
Ictericia

PLAN FOR THE DISTRIBUTION OF THE POLIOMYELITIS VACCINE UNDER THE NATIONAL VOLUNTARY DISTRIBUTION SYSTEM AND FOR ITS ADMINISTRATION UNDER THAT SYSTEM AND UNDER FEDERAL GRANTS THEREFOR*

I. RESPONSIBILITY AND AUTHORITY OF THE DEPARTMENT OF HEALTH.

In accordance with the recomendations of the National Advisory Committee on Poliomyelitis Vaccine and at the request of the Secretary of Health, Education and Welfare, the Governor of Puerto Rico designated the Department of Health, as represented by its Secretary, as the single agency to direct the distribution of the poliomyelitis vaccine in the Commonwealth of Puerto Rico.

The Department of Health of Puerto Rico is charged by law with the duty and responsibility of dealing with all matters which have to do with the health of the people. With relation to the distribution of vaccine his responsibility must be commensurate with the voluntary character of the system.

The Department of Health of Puerto Rico is authorized to receive from the Federal Government any and all grants intended to promote the health of the people and to administer programs under such grants subject to any applicable laws and regulations both Federal and of the Commonwealth.

II. BACKGROUND: THE EPIDEMIOLOGY OF POLIOMYELITIS IN PUERTO RICO.

Statistical data on the occurrence of acute paralytic poliomyelitis in Puerto Rico is available in the records of the Department of Health since 1928 when a small outbreak in a rural settlement was studied. Cases have been recorded yearly since 1930 when another small rural outbreak was studied. Island-wide epidemics have occurred in 1942 with 117 recorded cases (rate 6.0 per 100,000 population), in 1946-47 with 358 cases (rate 17.2 for the entire outbreak) and in 1954-55 with 501 recorded cases between November 1, 1954 and June 30, 1955 (rate 22.5 for the entire period). Otherwise, the year-by-year recorded cases and the case rates with relation to total population are as follows for the years indicated:

* Issued by the Department of Health of the Commonwealth of Puerto Rico.

Year	Cases	Case rate
1946	307	14.8
1947	57	2.7
1948	38	1.4
1949	52	2.4
1950	28	1.3
1951	89	3.9
1952	57	2.5
1953	31	1.4
1954	118	5.3

Generally, the epidemiology of poliomyelitis in Puerto Rico is that of poliomyelitis in tropical, underdeveloped countries:

(1) relatively low incidence of paralytic poliomyelitis with a tendency to gradual increase as socio-economic and sanitary progress is made;

(2) what paralytic poliomyelitis there is, in epidemic as well as in non-epidemic years, occurs at an earlier age than in highly developed communities;

(3) early natural immunity of a high proportion of the population, a theoretical deduction from (1) and (2) above, except for the fact that in 1930 McKinley and Soule (Proc. Soc. Exper. Biol. and Med., 1931, 29, 168) found neutralizing antibodies in the bloods of all of 8 unselected individuals.

The age distribution of 363 confirmed cases recorded between November 1, 1954 and March 25, 1955 is as follows:

Age Group	Recorded Cases	Per Cent of Total	Cumulative Percentage
Under 6 months	11	3.0	3.0
Six to 11 months	49	13.5	16.5
One to 2 years	193	53.2	69.7
Three to 5 years	71	19.6	89.3
Six to 9 years	27	7.6	96.7
Ten years or older	12	3.3	100.0

This age distribution is very much the same as that for the 1942 and the 1946-47 epidemic outbreaks and as that of the cases occurring in non-epidemic years. There is no noticeable tendency as yet for its increasing occurrence in the older age groups. During the 1954-55 outbreak the occurrence of cases in rural and urban settings has been exactly the same as the distribution of the general population, namely 52 per cent rural and 48 per cent urban.

III. COMBINED NFIP-COMMONWEALTH VACCINATION PROGRAM

Up to the time of the writing of this plan, no vaccination has been carried out under the NFIP program.

In accordance with the recommendations of official representatives of the American Medical Association, the American Academy of Pediatrics, the Association of State and Territorial Health Officers, the American Public Health Association and the U. S. Department of Health, Education and Welfare (Jan. 10, 1955) the NFIP did not originally allocate any vaccine to Puerto Rico. Upon inquiry, since Puerto Rico contributes annually to the NFIP funds through an organized chapter, the reason for the exclusion was later given as that "the pattern of epidemic poliomyelitis is considerably different from that experienced in the Continental United States and the territories of Alaska and Hawaii" and "it seemed inadvisable to extend the program outside of the United States and the territories of Alaska and Hawaii". Subsequently, the NFIP sent doctor Robert Ward, Professor of Pediatrics at the New York University Medical School, to look into the situation. Upon his recommendation, the NFIP decided to allocate 50,000 vaccines (then supposedly of 3.0 cc.) to Puerto Rico, to be administered to children 6 months to 2 years of age (children born between July 1, 1952 and December 31, 1954). There are in Puerto Rico 187,000 children in this age group (and 170,000 in the first and second grades in school plus some 35,000 who should be in those grades but are not in school).

On June 16 Parke Davis & Company and Eli Lilly and Company contracted to supply the Government of Puerto Rico 17,846 vials of vaccine of 9.0cc. each as soon as marketing conditions permitted.

Neither the NFIP nor Parke Davis & Company and Eli Lilly and Company have delivered this vaccine. When we have all of it on hand (all three deliveries), we intend to go ahead with the vaccination of this group, hoping to reach about 80 per cent of the total. The Department of Health is ready for this task and will undertake it within two weeks of the receipt of the total vaccine expected for this combined NFIP-Commonwealth Program, specific plans for which are as follows:

For one entire week every public health unit in the Commonwealth, one in each town, will dedicate itself exclusively to this vaccination (except that communicable disease control, —including ambulatory treatment of tuberculosis— and slaughterhouse daily inspections will be continued). The first injection will be administered to 130,000 to 140,000 children in this age group during this week. Four (4) weeks later every public health unit will

again dedicate practically its entire personnel and time to the task of administering the second injection. Education to this end is now going on in every municipality, and it will be intensified as soon as the exact date is set on which vaccination is to begin.

The necessary equipment is on hand and the personnel of every health unit has been trained in the details of record keeping, the administration of the injection, etc.

Proof of age will be expected but not required except in case of great doubt (every child in this age group has been given a certificate of registration at the time of birth registration). No inquiries are to be made concerning economic status.

IV. TENTATIVE CONTINUING COMMONWEALTH VACCINATION PROGRAM WITHOUT FEDERAL AID.

The Legislature of Puerto Rico has included in the appropriations for the Department of Health, fiscal year 1955-56, the sum of $200,000 for the purchase of poliomyelitis vaccine. We had planned to administer the third dose to those included in the combined NFIP-Commonwealth Program about 7 months after the first injection. We do not know when this might fall due since we do not know when the NFIP vaccine will be delivered, nor when Parke Davis & Company and Eli Lilly and Company will deliver the vaccine they have contracted for.

We had planned to vaccinate as many as possible of the children in the 3 to 5 year old group, the group with the second highest susceptibility; this group represents a total population of 232,000. We also planned to vaccinate before next Spring, in March and April, (2 injections) all children who had reached the age of 6 months (born between January 1, 1955 and August 31, 1955) some 40,000 children.

(1) The administration of the third injection to the children vaccinated under the combined NFIP-Commonwealth Program would be done again by every public health unit devoting one entire week almost exclusively to this task.

(2) The vaccination of the approximately 40,000 children who had reached the age of 6 months by March 1956 (born between January 1, 1955 and August 31, 1955) would be done by each public health unit devoting two (2) full days of March and two (2) full days of April to this activity. It is estimated that we can vaccinate about 26,000 children daily throughout the entire Island.

(3) The vaccination of the 3 to 5 year old would be done by each public health unit dedicating one day, or part of one day, each week to this activity, except during the weeks when groups (1) and (2) were being vaccinated.

The appropriation for this program was $200,000 for the fiscal year 1955-56, to be applied approximately as follows, at current prices of the vaccine ($0.65 per cc.).

(1)	For third injection to group (1), 140,000 children at one injection each	$90,000.00
(2)	For vaccination of group (2), 40,000 children at 2 injections each,	52,000.00
(3)	For vaccination of approximately 45,000 of the children in group (3),	58,000.00

It had been planned to obtain a yearly Commonwealth appropriation of $200,000 with which each year we would vaccinate approximately 60,000 children which would reach each year the age of six (6) months ($120,000.00) and about 40,000 yearly of the unvaccinated $80,000.00) in the now 3 to 5 year old, expanding the elligible group upwards to include those reaching the age of 9 years (not quite the tenth birthday) whenever they were reached.

It was felt that a large-scale vaccination program in children over 10 years of age was not justified under present circumstances. No case of poliomyelitis has been registered in Puerto Rico in a pregnant woman, which is perhaps due to the fact that probably natural immunity at the age of 15 is quite universal.

V. TENTATIVE COMMONWEALTH VACCINATION PROGRAM WITH FEDERAL ASSISTANCE.

1. The uncertainty that there is even today concerning the date on which any vaccine will be available to Puerto Rico and of the amount of vaccine which will periodically be available before the manufacturers reach maximum production makes it quite impossible to formulate any definite plans of action even with the expectation that federal assistance will be available.

2. Under the circumstances, we should go ahead with the combined NFIP-Commonwealth Vaccination Program as planned and as described in III above soon after Parke Davis & Company and Eli Lilly and Company deliver the vaccine for which they have contracted. Parke Davis & Company should be enabled to make this delivery during the first month of vaccine being available; 1954-1955 fiscal year funds have been obligated and this purchase cannot be split at this time.

3. The federal allotment of funds to Puerto Rico is calculated to provide enough vaccine for 41 per cent of the population up to 20 years of age. If it should be accepted that large-scale vaccination is justified only up to age 9, the total federal assistance allot-

ment would be sufficient to vaccinate 100% of the population up to 9 years of age (or very close to 100 per cent) which would be approximately 41 per cent of the population under 20 years of age. Sec. 4 (a) of Senate Bill 1948, as amended (to June 14) seems to preclude the possibility that the total federal grant can be so used.

4. Any realistic program for vaccination against poliomyelitis in Puerto Rico should be based on the following order of priorities based on relative susceptibility of age groups:

1st priority, children 6 months to 2 years of age
2nd priority, children 3 to 5 years of age
3rd priority, children 6 to 9 years of age
4th priority, children 10 to 20 years of age.

5. The third injection shall be given to the participants in the combined NFIP-Commonwealth Program during one week to be dedicated to this purpose by every public health unit 7 months after the first injection. This will complete the vaccination of the First Priority Group in the course of the current fiscal year if the first injection can be administered no later than September.

6. During two (2) days in March and two (2) days in April, 1956, all public health units will devote their entire personnel and effort to vaccinating (first and second injections) children who have arrived at the age of 6 months and have not been vaccinated as under 2, this section.

7. For the vaccination of the Second Priority Group, as well as for the vaccination of First Priority Group, children who did not come for vaccination during the development of the combined NFIP-Commonwealth Program as under 2, this section, every public health unit shall set aside one day every two weeks, the same day for all of them throughout the Island and to be designated by the Secretary of Health. The weeks on which 5 and 6 above, this section, are to be carried out shall be excluded, as also any week on which the designated day is a legal holiday. If federal funds are available, and sufficient vaccine, this phase of the Program can be initiated very soon after the combined NFIP-Commonwealth Program (2 injections) is completed. Because of uncertainty in this respect, it is impossible to estimate how many children will be vaccinated in the course of the current fiscal year, but assuming that in the course of one full calendar year 22 such days can be devoted to this activity, at a capacity of 26,000 children (injections) per vaccinating day, 286,000 can be vaccinated (2 injections) in that length of time besides whatever number is reached by the combined NFIP-Commonwealth Program with some 187,000 potentially elligible. This signifies 473,000 may have been vaccinated by December

1956, and probably represents the population up to 10 years of age. No plans should be made for vaccination of the Fourth Priority Group prior to 1957. (During 1957 there would be, besides the complete vaccination of those arrived at 6 months of age, the administration of the third injection to some of those who receive the first 2 injections during the previous calendar year.)

8. During the period during which any priority group is being vaccinated, every elligible child in that group will be offered the opportunity of being vaccinated, irrespective of economic status or place of residence; also those in priority groups previously offered the opportunity to get vaccinated if they did not at the time avail themselves of the opportunity.

9. The entire facilities of the Department of Health for education of the public will be used to assure participation of the largest possible number of each priority group. This includes the mobilization of the community as such and the assistance of other governmental agencies —the school system, the Agricultural Extension Service of the University of Puerto Rico, the Social Programs organization of the Department of Agriculture, the Community Education Division of the Department of Education, etc. It also includes the use of all media and techniques for such education.

10. If the demand for vaccination in any priority group after the First Priority Group exceeds the 41 per cent which is covered by the federal grant within each priority group, Commonwealth funds will be available for the vaccination of any children demanding vaccination in excess of the said 41 per cent within that priority group.

11. The above program provides for the vaccination of the population to (approximately) age 10; furthermore, it covers practically the entire period during which the federal grant allocations is available. It is now felt that, in the light of the nature of the paralytic poliomyelitis situation in Puerto Rico and in the light of the urgency and overcrowding of other public health programs, no more time and effort of the public health units should be dedicated to this program than is herein indicated. Since it implies that not all of the federal grant allocation will be used, the opportunity is requested to submit, as experience may dictate, the necessary amendments in the near or more distant future.

12. It can be anticipated that some children within each priority group will be vaccinated by private practitioners rather than through the governmental program even if the opportunity to be vaccinated within the governmental program is offered to all irrespective of economic status. It might be estimated that about 20 per cent of the total will be vaccinated through the private practice of medicine after private practitioners and their clients

regain confidence in the vaccine; not before. (Seventy to 80 per cent of families are considered to be medically indigent on the basis of having less than $2000 annual income per family, but many of these prefer to go to private practitioners and buy their own medicines).

VI. PLAN FOR DISTRIBUTION

A large demand can probably not be expected from private practice and the direct purchase of the vaccine; not at least in the early days of distribution. On or about April 15 one manufacturer distributed 7,000 ampules directly to physicians and to drugstores. The distribution to drugstores was fair roughly proportionate to population of municipalities; it can be said that it was probably available to anyone who wanted to buy it. There was no binding agreement that it could be prescribed or sold for any particular priority group and it is well known that it was administered to children of all ages and to adults. On May 24 all drugstores were asked to report on the number of vials they had on hand; on June 22, one month later, a random sample of 29 drugstores throughout the island reported they still had on hand 89.6 per cent of the vaccine they had on 24 May.

All 6 manufacturers of the vaccine, except one, distribute all their products from stock maintained in Puerto Rico by their direct representatives, orders from whole-salers and from retailers being received by them. The one exception (Eli Lilly and Company) has a representative in Puerto Rico who maintains no stock; orders may be sent to him for transmittal to the factory or may be sent directly to the factory by five whole-sale distributors. In no case are deliveries made directly from the factory to the retailers.

Up to the time of the writing of this plan, none of the manufacturers' representatives has received any instructions from their concerns, nor any notice of the agreements they have entered into for distribution at the national level. They are requesting information and instructions from their respective concerns, but foresee difficulties ahead in the plans for distribution which are prescribed at the national level.

(1) They foresee difficulties in by-passing the whole-salers, an integral part of the usual, normal, marketting process. The 12 whole-salers in the island have no organized association at the moment, hence can not be represented in the Advisory Committee. They will have to be approached individually for agreement on the general policies of distribution and reporting if they are not to be left out. Manufacturers' representatives have no instructions from their companies to by-pass them.

(2) Copies of invoices to retailers will have to come from whole-salers if they participate in the distribution system; the manufacturers or the manufacturers' representatives will send copies of invoices as deliveries are dispatched to whole-salers. The manufacturer's representatives question whether there is a legal basis for stopping deliveries to retailers because of non-compliance with agreements on reporting by them. The manufacturers' representatives also forsee difficulties if physicians specify in their prescriptions the particular manufacturer whose vaccine they wish to be dispatched.

(3) There are 556 drugstores in Puerto Rico, some 400 of which are members to the P. R. Retail Druggists Association. The Association is not affiliated to any similar national association but commits its members to the agreements. The Pharmaceutical Association (membership of all registered pharmacists is compulsory by law) commits its members to the agreements.

(4) The manufactuers' representatives agree to the general policy of distribution to which their concerns have agreed at the national level, subject to eventual decisions concerning the participation or non-participation of whole-salers in the distribution system.

(5) Initially, 20 per cent of the monthly supply of vaccine will be tagged for distribution to the retail business through drugstores (surgical supply houses do not distribute biologicals in Puerto Rico). Eighty (80) per cent of the monthly supplies in Puerto Rico will be tagged for purchase by the Government of Puerto Rico in accordance with available funds and the month-to-month demands of the vaccination program.

(6) As the allotment of vaccine for Puerto Rico for the following month is known, a determination will be made as to the amount thereof to be purchased by the government within the 80 per cent allocated to the public vaccination program. Drugstores will be immediately requested to signify the number of vials they are desirous of obtaining for the following month. The Department of Health, in agreement with the manufacturers' representatives, with or without the concurrence of the whole-salers depending on whether they participate or not, will distribute amongst them the vaccine available to the retail trade within the 20 per cent alloted to it. If in any one month on which the Government is not to purchase its entire allotment there is a demand for the retail trade in excess of the 20 per cent alloted to it, the necessary adjustments will be made so that as much goes into the retail trade as there is a demand for and subject to the determination, in as far as it can be determined, that the vaccine is being administered only to the age group for which at the time it is intended. The

Public Health Service will be notified of the total to be absorbed out of the allotment of that month to Puerto Rico, and what proportion of it is to go to the retail trade and how much to the Governmental Program.

(7) There are 1,200 physicians in Puerto Rico, of which 700 are members of the Puerto Rico Medical Association which is an affiliate of the American Medical Association. The Puerto Rico Medical Association pledges its members to abide by the agreements on which the voluntary control system is predicated.

(8) The manufacturers' representatives, for the manufacturers, working through their distributors or not through them, agree to:

(a) Abide by the tentative 80 per cent allocation of the monthly Puerto Rico quota of vaccine to the government and 20 per cent to the retail trade, subject to the changes thereof which public demand in the retail trade, the needs of the public programs and the availability of the vaccine may justify, the actual percentage to be decided upon by the Secretary of Health of Puerto Rico in consultation with the Advisory Committee.

(b) Send a copy of each invoice, whether delivery is made to whole-salers, retailers or physicians, to the Secretary of Health of Puerto Rico on the same day on which delivery is dispatched; if whole-salers participate in the distribution system, they are to —in turn— submit to the Secretary of Health one copy of each invoice on the day on which every delivery is made.

(c) Enclose if they make the deliveries themselves, or come to be enclosed if delivery is made by whole-salers as distributors, in every delivery to retailers, sufficient copies of the printed form of the Public Health Service now known as Form C —"Report on Retail Pharmacy or Surgical Supply House on Poliomyelitis Vaccine Sold".

(d) Distribute the vaccine available to the retail trade to drugstores throughout the island in accordance with the demand in the light of the vaccine available and as decided upon by the Secretary of Health.

(e) Withhold delivery of vaccine to any whole-saler, retailer or physician who does not comply with the terms of the agreement, subject to the limitations that the voluntary character of the distribution system imposes.

(9) The Pharmaceutical Association (College) of Pharmacists and the Association of Drugstore Owners bind their members to

(a) Sell no vaccine except under prescription from a duly licensed physician, in accordance with the law.

(b) Make a weekly report of sales to the Secretary of Health of Puerto Rico, in the form now known as Form C of the Public

Health Service, "Report of Retail Pharmacy or Surgical Supply House on Poliomyelitis Vaccine Sold" which will be included by the manufacturer or distributor with every delivery of the vaccine.

(10) The Puerto Rico Medical Association pledges its members to

(a) Vaccinate or prescribe vaccine only for children in the age group for which vaccination is intended at the time in accordance with the approved Plan except in exceptional meritorious cases.

(b) Keep accurate records of the cases vaccinated by them through their private practices including name and address, date of birth, dates of injections, sites of injections, name of manufacturer and lot number of each injection and volume thereof, untoward effects.

(c) Not to stock-pile vaccine in excess of their actual needs for vaccination of children within the age group being vaccinated at the time.

(11) The Department of Health of Puerto Rico pledges itself to administer the plan of distribution of vaccine within the limitations imposed by its voluntary character and nature by:

(a) Setting up an Advisory Committee, already appointed, which consists of

Mr. Francisco Castagnet, Puerto Rico representative of Wyeth Laboratories, representing the representatives in Puerto Rico of the six (6) manufacturers of vaccine.

Dr. Guillermo Arbona, Professor of Preventive Medicine and Public Health and Head of the Department, School of Medicine of the University of Puerto Rico.

Dr. Alberto Díaz Atiles, Chairman of the Section of Pediatrics, Puerto Rico Medical Association, representing the Puerto Rico Medical Association.

Dr. Egidio S. Colón Rivera, Puerto Rico Chairman of the American Academy of Pediatrics.

Mr. Esteban Amador, President of the Pharmaceutical Association (College of Pharmacists) in representation thereof.

Mr. Santos Feliú, President of the Puerto Rico Retail Druggists Association (drugstore owners whether pharmacists or not) in representation thereof.

This Committee as a whole will advise on matters pertaining to distribution of vaccine and related aspects. The three physicians will constitute an Advisory Technical Subcommittee on technical matters pertaining to age group priorities, the public vaccination program, techniques, etc.

(b) Set up the administrative staff and organization within the structure of the Department of Health, if federal funds are available, for handling matters pertaining to distribution.

(1) Receiving invoices from manufacturers or distributors
(2) Receiving reports from drugstores
(3) Establishing allocations for the retail trade in the various municipalities.
(4) Keeping track of vaccine distributed to municipal jurisdictions through public vaccination program.
(5) Setting up system for detailed statistics on vaccine distributed to the municipal jurisdictions as the area units for distribution of the vaccine in relation to number of children in the priority groups being vaccinated successively. Prepare forms therefor.
(6) Prepare reports for the Public Health Service and/or the National Advisory Committee.

(c) Establish clinics in public health units and subunits —urban and rural, as necessary, to assure that the vaccine and its injection is in an orderly fashion available to children in the designated priority groups who are not otherwise vaccinated privately and have vaccination requested by their parents or guardians.

(d) Make every possible effort to have all groups concerned, as well as the general public, adhere to the orderly fashion in which the vaccination is to be carried out according to the priority groups.

(e) Carry out, with the assistance of other public and private agencies, the educational campaign that will assure participation by the largest possible number of the elligible amongst the successive priority groups.

(f) Watch for iniquities in the distribution of vaccine available, either in the retail trade or in the public vaccination program, correct them whenever possible and report to the U. S. Public Health Service as necessary.

ASAMBLEA ANUAL

Diciembre 6-10, 1955

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Agosto 26 y 27, 1955

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O LAS RADIOEMISORAS DE
PONCE Y MAYAGUEZ

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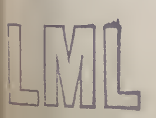
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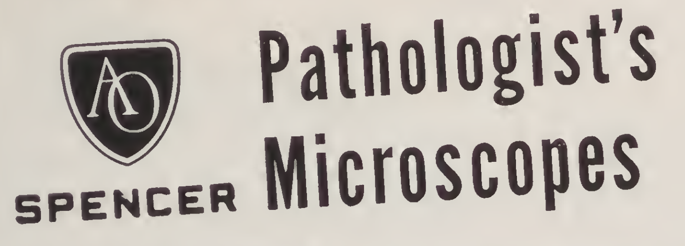
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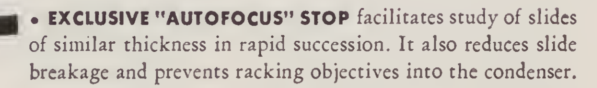
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BOLETIN

DE LA ASOCIACION MEDICA DE PUERTO RICO

VOL. 47 SEPTIEMBRE, 1955 No. 9

ACCESSORY INTRAPLEURAL ECTOPIC LIVER LOBE

DAVID RODRÍGUEZ-PÉREZ, M.D.
Rio Piedras, P. R.

Intrapleural accessory liver most be a rare finding from both, the anatomical and pathological points of view. In the literature reports are made of ectopic liver or lobes in the chest, but in reading the reports, it is easy to discover that the publications concern herniated lobes or liver thru one or the other diaphragmatic leaf. The author wants to make it clear, that in this report we are dealing with a case of ectopic liver tissue, totally independent in the right pleural cavity with the bile duct, portal system, lying over an intact diaphragm. The pedicle of the liver pierces thru the diaphragmatic cupple into the abdominal liver so that, on deflation of the lung, the liver seems just to "sit" over the right diaphragm, moving freely in the chest and hanging only from its rather small pedicle.

In reviewing the literature it has been impossible to find the above described picture in the human. We find only a description of this type of ectopic liver in the cow, as described in 1889 by Guinard[1] who was Sanitary Inspector at Dijon. Before this, all reports on animals and man were simple diaphragmatic herniations of the liver.

In 1925 Thomas S. Cuellen[2] from Hopkins, after finding a small accessory hepatic lobe springing from the gall-bladder, made the most exhaustive study of the subject. He reviewed the literature up to his time, but such a case as we are reporting here was not found by this author.

We turned to the Reference Division of the Armed Forces Medical Library and they kindly reviewed the Quarterly Cummulative Index Medicus as well as the Excerpta Medica and Current list of Medical Literature. We personally searched the Index Catalogue of the Library of the Surgeon General's Office and all the references sent to the author as well as the personally studied references lack to reveal a parallel case to the one being reported.

REPORT OF A CASE

Case C. G. Unit No. 116179 - Bayamón Dist. Hospital.

The patient is a 15 year old white girl who on a routine X-Ray was found to have a mass in the right lower chest in the supradiaphragmatic region. For this reason was sent to us for diagnosis and treatment. The physical examination as well as the laboratory data were essentially within normal limits.

X-Rays were taken again. The reading by our Radiologist is as follows:

"Chest: there is noted a relatively round mass in the supradiaphragmatic area of the right chest laterally and posteriorly in position. Its borders are smooth and there is no evidence of calcification in this mass which measures 6 cms. in its greater diameter. The border of the diaphragm is sharply delineated and the lower border of the mass follows its contour suggesting separation from the diaphragm but a fairly collapsible mass (cyst). The density of this process is about the same as the cardiac density. The examination is otherwise negative.

Conclusion: Mass at the lower lung field, laterally and posteriorly. Bronchogenic (lung cyst) must be considered in the diagnosis. Fluoroscopic examination is recommended."

Operation: On the 29 of October, 1954, patient was submitted to an exploratory thoracotomy. Incision was done between the 7th and 8th ribs. The thorax was entered thru the intercostal space. Small posterior segments of the 7th and 8th ribs were resected. Finochietto's retractor applied. Chest explored - mass found free from adhesions. The vascular pedicle of the mass was doubly ligated, cut, and removed. At the pedicle a 1 cm in diameter portal vein and 3 mm. in diameter bile duct could be well dissected. The arterial supply was mainly thru a very small artery. The post-operative diagnosis of ectopic liver lobe was confirmed by hystological studies of the specimen.

The patient was discharged on 11/7/54, 8 days after operation.

She had an uneventful recovery. Lung reexpanded well and wound healed **"per primam"**.

Patient has been back to school, comes regularly to OPD clinic.

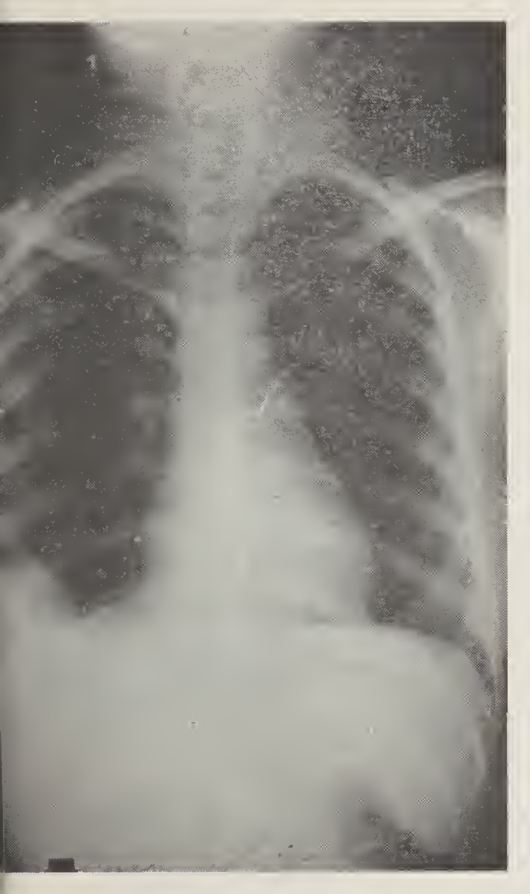


Fig. 1

Pre-op. X-Ray PA view.

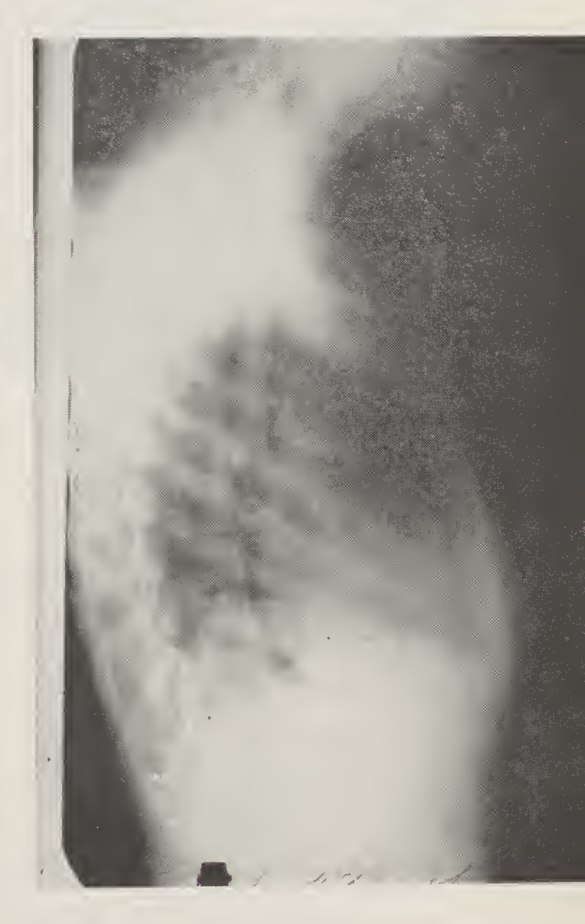


Fig. 2

Pre-op. X-ray lateral view.

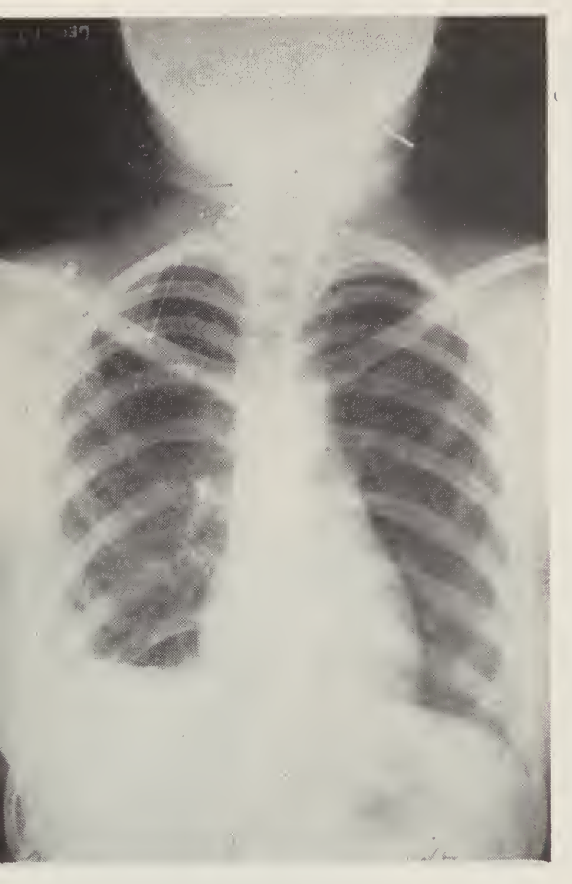


Fig. 3
Post.-op. X-ray showing excellent reexpansion of lung.

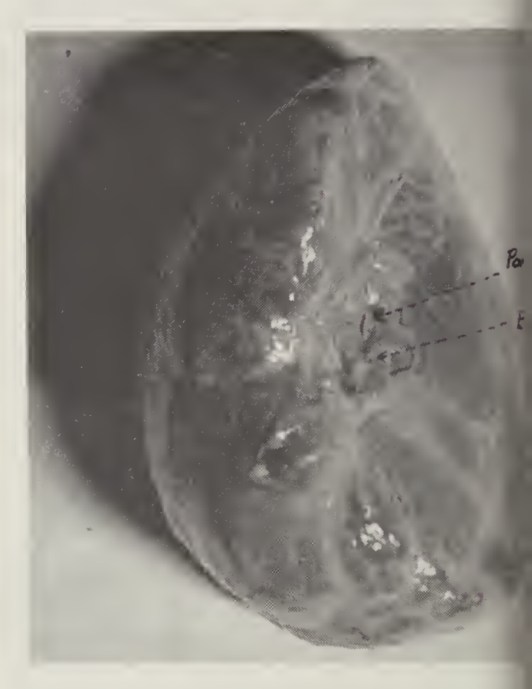


Fig. 4
Liver lobe showing diaphragmatic surface pedicle structures of liver lobe.

SUMMARY

A case of intrapleural accessory ectopic liver lobe is reported, apparently the first case occurring in the human as reviewed in the world literature.

SUMARIO

Se informa un caso de lóbulo hepático ectópico accesorio localizado en la cavidad pleural, aparentemente el primer caso que se informa en el humano después de repasar la literatura mundial.

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INFANTILE CORTICAL HYPEROSTOSIS*

REPORT OF ONE CASE

*JUAN BASORA DEFILLÓ, M.D.***

*JOSÉ LANDRÓN BECERRA, M.D.****

J. R. is a white female baby, 3 months old. She was delivered normally weighing 7 lbs. 5 oz. The baby was given an evaporated milk formula and vitamins, and continued well until 2 months of age. The baby began to cry and moan frequently, episodes which the mother described as colics. The formula was changed to cow's milk but within a week it was again changed using goat's milk assuming that the symptoms were allergic in origin. The baby did not improve. She continued with colics, anorexia. When we saw her at 3 months of age she was fretful, irritable and pale. She had fever, pain over the body and would cry on slight movement. The head was dropped backward and there was some neck rigidity. She was hospitalized for observation.

Laboratory report: Hbg. 8 gms. RBC 3,000,000, WBC 15,400. N 47 per cent, L 48 per cent, E 2 per cent, monocytes 3 per cent. The neck rigidity persisted, so a spinal tap was done, which yielded normal results. Blood serology was negative. Blood transfusion of 100 cc. whole blood was given and she was returned home very little improved after 3 days in the hospital. The child continued sick and this very same day it was noted that she had swelling over the right side of the thorax. The soft tissue swelling was tender, but not fluctuating. The baby was given Terramycin and large doses of ascorbic acid. X-rays of the chest were taken and reported as follows: "Examination of the chest shows considerable cortical reaction of the right 3, 4, 5, 6, 7th ribs. These findings are consistent with infantile cortical hyperostosis." (Fig. 1). X-rays repeated 2 weeks later showed cortical reaction in the left clavicle and in the 8, 9, 10th left ribs, (Fig. 2). X-rays of the affected bones taken one year after showed complete regression of the cortical reaction, (Fig. 3). This child is now 4 years of age and has grown and developed normally.

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*Presented during the Annual meeting of the P. R. Medical Assoc., Dec. 10, 1954.

** Director Dept. of Pediatrics, Municipal Hospital, Río Piedras, P. R.

*** Director, Dept. of Radiology, Presbyterian Hosp., San Juan, P. R.



Figure 1 — X-ray taken Sept. 1, 1951. Note cortical reaction of right 3, 4, 5, 6, 7th ribs.



Figure 2 — X-ray taken Sept. 17, 1951. Note additional cortical reaction now involving the 8th, 9th, 10th left ribs and the left clavicle.



Figure 3 — X-ray taken October 8, 1952. Shows complete regression of the cortical changes previously described.

Discussion

Infantile cortical hyperostosis occurs mainly in children under six months of age, but some children with late onset in the second year have been reported. This disease has an obscure etiology and pathogenesis, but there is increasing evidence of an intrauterine or genetic origin. This syndrome was first described by Caffey and Silverman in 1945.

The principal symptoms and signs described in Caffey's original report[1] and in several others which have appeared in the literature are irritability, tender soft-tissue swellings, and roentgenographic cortical thickening of the underlying bones. Burke et al[2] are under the impression that the skeletal lesion is primarily a periosteal reaction rather than a cortical involvement. The tender soft tissue swellings are deeply situated and the skin over them is never warm and does not show any discoloration and is movable. They appear suddenly clinically and are noticed before the cortical thickening becomes visible roentgenographically. They disappear long before the X-ray lesions completely recover. They involute slowly without suppuration. Areas of tissue swellings have been seen to recur suddenly in the original sites or may appear in new sites during the course of the disease. Fever is a constant finding. Other clinical findings described are: extreme pallor, painful pseudoparalysis of the extremities and pleuresy. Painful swelling of the mandible is a common feature in infants.

Caffey[3] states that "for some unknown reason, facial and scapular swellings have never first appeared after the six month of life." The sedimentation rate is elevated and there is increased phosphatase. Leucocytosis is present in almost all the cases. Serology tests are negative. Cultures made from tissues of these children are negative.

The cortical lesions in the bones, which is the main characteristic of this disorder, have been shown in all the tubular bones of the skeleton, the flat bones, the mandible, parietal and frontal bones, and the scapula. The scapular lesions are reported as always been unilateral and always appearing under six months of age. Clavicular lesions may be unilateral or bilateral and may be present in all ages. Whipple[4] described a case with involvement of skull and bones of the calvarium. However, changes of cortical nature do not occur in all the bones in the same patient. The roentgen lesion is one of cortical thickening in the bones. Lamination may occur during the healing process. Caffey states that he has never seen lamination early in the disease. Smyth et al[5] described 7 cases in which the periosteal reaction producing new bone gave a laminated onion peel appearance. Bennett and Nelson[6] report X-rays of a fetus in utero that showed marked thickening of both radii and ulnae.

The disease runs a mild course. Clinical and roentgen recovery is complete in most patients. Deaths have been reported recently. Late skeletal change have been described by Caffey[7] in a negro girl producing deformity and crippling. Caffey states: "Hyperostosis are usually invisible within 12 months after the swelling in the soft tissue and the fever have subsided." The course of the disease is not modified by administration of antibiotics, or by the administration of large doses of ascorbic acid. Sidbury and Sidbury[8] feel that ACTH and cortisone shorten the course of the disease and prevent residual bone deformity. They recommend hormonal therapy in the more severe cases.

Differential Diagnosis

The disease has been confused with scurvy. In scurvy there is a history of lack of citrous fruits in the diet or a diet exclusively on pasteurized or boiled milk. Scurvy occurs in infants 3 months of age or older. There are no authentic cases of symptomatic or roentgen evidence of scurvy in infants younger than 3 months of age.

Generalized sarcomatosis may be mistaken for it. The abnormal

picture of the bones with swelling of the cortices and lamination, may lead one to suspect sarcoma.

Multiple osteomyelitis may be misdiagnosed to the extent that some patients have been reported erroneously operated. In osteomyelitis, the systemic symptoms are severer and there is a response to antibiotics.

Congenital syphilis can be ruled out by a negative serology. The X-rays of the bones in infantile syphilis show lesions that involve the diaphysis and the metaphysis of each of the several bones affected.

In tuberculosis of the bones, there is a positive tuberculine test. The lesions are hematogenous in origin and usually single. The bones most frequently involved are the femur, the vertebrae, the fingers, and the toes.

Traumatic periostitis of the newborn may be misdiagnosed and mistaken for cortical hyperostosis. There is a history of fall before onset of illness. Injury to the bones producing multiple fractures may result in a periosteal reaction, which can be confused with a cortical reaction.

Fever, pain, and tenderness over the extremities, increased sedimentation rate may point to rheumatoid arthritis or to rheumatic fever.

Anemia, fever, and bone pain are seen in leukemia. The blood picture and bone marrow studies in leukemia will clear the diagnosis.

Parotitis may be misdiagnosed due to the facial swelling.

Hypervitaminosis A has been described in some children and infants who have ingested large amounts of concentrates of vitamin A. These children are irritable. Their temperature is not elevated and they have tender soft tissue swelling. The blood vitamin A determination is elevated over the normal of 50-100 gms. There is cortical thickening of the bones.

Summary

We have reviewed the symptoms, pathogenesis, and X-ray findings in infantile cortical hyperostosis. A case of this interesting disease, in a baby girl, three month old, has been described.

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TUBERCULOSIS CONTROL ACTIVITIES OF THE HEALTH DEPARTMENT

*HAROLD HEIMAN, M.D.**

For a meeting of this type, I would have preferred to have had several weeks in which to prepare all the information I would have liked to present. But the time was not available, and therefore my talk will be based on some thoughts that I recorded on a sheet of paper. From this I will tell you about what I think of the present status and, from that, what the future looks like in tuberculosis in Puerto Rico.

The mission of the Bureau of Tuberculosis of the Division of Public Health of the Department of Health is to act as the focal agency for the prevention of the spread of tuberculosis. Information about all things which are done for the prevention of tuberculosis in Puerto Rico would come to our bureau. We are the focal point of tuberculosis control in the island. That is the way we see our mission.

But, let us at once recognize that we do not, of course, do all the work ourselves for the control of tuberculosis in the island. We couldn't possibly do it all. There are many other groups in the Department of Health, in the Division of Public Health, and other parts of the Insular Government which do work for tuberculosis control.

Among these are the following: The Division of Hospitals, the Bureau of Health Education, the Bureau of Laboratories, the Bureau of Vital Statistics, the Bureau of Public Health Nursing, the Division of Public Welfare, and the Bureau of Transmissible Disease. All are working in the direction of the control of tuberculosis on the Island.

Now our responsibility is to the Secretary of Health, through the Director of the Division of Public Health. The basic way in which our Bureau does its work is by keeping records of cases of tuberculosis, and analyzing those records, so that we may see where and what kinds of work need to be done. Our Bureau exercises technical control only of our Tuberculosis Centers, scattered all over the island, and we have technical control and direct control of our three mobile x-ray units.

What is the present status of Tuberculosis here? At the moment, we estimate that there are over 10,000 **administratively** active cases attending our Tuberculosis Centers. Many of you have

* Director, Bureau of Tuberculosis of the Division of Public Health of the Puerto Rico Department of Health, San Juan, P. R.

heard of our efforts to establish new case registers for each of our Tuberculosis Center. This work is in progress, and has been so for the past 9 months. When these are all established we shall then have accurate information, and not estimates, of the numbers of cases.

The second point I should like to make in regards to our present status is that we are short of technical people. We are short of physicians and we have a hard time getting physicians. We suspect that we do not pay them enough; but that is what we can pay, and we therefore have a hard time getting doctors. Now, we are not only short of physicians, but we are short of nurses.

The physical facilities of our Tuberculosis Centers need much to be improved and are being improved, slowly.

Now, about the recent changes that have occurred in our Tuberculosis control activities.

We have had a drug program for some time, but only since October 1952 did we include large numbers of people in our drug program. Every patient on the island who has tuberculosis can get the drugs, the effective drugs, Streptomycin and Isoniacide. We estimate that over 12,000 ambulant patients have entered the drug program.

In 1952 our tuberculosis death rate was 94 per 100,000. In 1953 it was 47 per 100,000 a drop of 50%. When we saw that drop of 50%, we were quite worried of what was probably going to happen in 1954. We thought at the time, that those patients whose lives we were saving in 1953 would probably die in 1954. They didn't. In 1954 we continue to have a drop in tuberculosis mortality to 38 per 100,000, that is a 17% drop over what we had in 1953.

Now about the morbidity data. Morbidity data is based entirely upon good case reporting. We think it is fair in the island; but we also think that it can be improved. We believe that it is relatively stable.

In 1952 we had 216 active and probably active new cases reported per 100,000 of the population. In 1953, it was 186 per 100,000, a drop of 13.8%. In 1954 we have 152 per 100,000, a drop of 18% over that of 1953. The trend there is really remarkable. Again, you have the typical curves when you compare mortality drops with morbidity drops, curves which you have seen compared all over the world; that mortality is dropping fast, morbidity much more slowly. But morbidity, we think, is dropping quite well. But there are a few other things to say about morbidity rates. In analyzing our data, (1953 and 1954), and comparing them with 1952, we find that we are getting earlier reporting of cases as judged by extent of disease.

We are also finding other significant things in the data for these 3 years. The decline in new cases is apparently occurring especially amongst those between 20 and 44 years of age. It seems that more cases are occurring in the older people and less in the age group 20 to 44.

We like that idea; we like the idea of getting our cases reported early; we like the idea that people between the ages of 20-44 are having less tuberculosis.

We find another very encouraging point; more patients in 1954 and 1953, than in 1952, came to our Centers for symptoms than they had done before. In the earlier years they came mostly for routine health certificates. Now more of them are appearing for symptoms. Again, this is a good trend.

Recently we had an opportunity to study some Tuberculin Sensitivity data, as a measure of infection rate.

From tentative data that I have, it appears that for children, born a few months after October 1952, when we started our intensive drug program an appreciably lower incidence in the positive Tuberculin Sensitivity occurred than in the same age group born before October 1952. These data suggest that there is a lowered infection rate in these children.

We are just now completing the collection of data of Tuberculin Sensitivity of first-grade school children done on a random basis over the island. We find that when we compare the data for this year with that which we have for 1949 to 1951, it appears that there is a drop in the Tuberculin Sensitivity rate amongst the school children of that age group; especially so in the urban areas, rather than in the rural areas.

We made an analysis of 1,000 consecutive cases of tuberculosis that were receiving drugs at the Centers close to San Juan. We read the chest x-rays before and after therapy had started. We found that 50% showed remarkably good roentgenographic improvement. They showed excellent improvement in spite of the conditions in which many of our ambulatory patients are being treated. Many of them get their injections at home, from some member of the family or friends and others get their Streptomycin right at the Tuberculosis Centers. The drugs that are used in our tuberculosis program, in the way that we are using them, are quite effective as judged roentgenographically.

That briefly is our story I wanted to tell you a little bit of what our Bureau is, how we function, and what we are doing. I wanted also to tell you about our mortality and morbidity data and their recent changes. I leave it to you to project into the future and see what is going to happen to Tuberculosis in the island.

EL HOSPITALISMO MENTAL*

PEDRO MENDOZA MENDOZA, M.D.

Hace 12 años mi profesor Eugenio Cienfuegos nos enseñaba que cuando un niño llevaba varias semanas hospitalizado y su condición física estaba detenida o tendía a empeorarse, había siempre que considerar el concepto Hospitalismo, que significaba falta de cariño maternal, recuerdo de su hogar por muy humilde que este fuese. Este concepto no era nuevo. A principios de este siglo la Escuela Pediátrica Alemana con Pfauldler y Finskeltein después, y la Francesa con Marfan, ya lo habían preconizado. Y este concepto clínico de observación empírica era tenido muy en cuenta por pediatras de ese entonces. Hospitalismo expresado en una baja de la salud en general, gran propensión a las infecciones intercurrentes, tendencia acusada a bajar de peso, irritabilidad y anorexia, a pesar de los cuidados más solícitos prodigados, ellos reaccionaban de una manera negativa, por lo que era más prudente darles de alta y en muchas oportunidades, observábamos una reacción paradojal, el niño en su hogar y con su madre recuperaba el apetito, dormía mejor, y a veces curaba de sus infecciones. El profesor Cienfuegos nos decía que se debía a una "carencia afectiva", aunque por otro lado era negado, el Dr. Juan Garrahan de la Argentina, nos refería que ello se debía a las malas condiciones de higiene y cuidado de los niños en Hospitales y Asilos. Pero con el adelanto de la Medicina Infantil, con el trabajo en equipo del Psicólogo y el Psiquiatra de niños, este concepto tiende a evolucionar y hacerse más científico en su interpretación, denominándose, por ahora Hospitalismo Mental y en el cual por medio de estadísticas y observaciones clínicas se demuestra el Trauma psíquico que significa la separación brusca del niño de su hogar, injuria mental que se acentúa según el carácter del niño y el tiempo de permanencia en el Hospital.

Recientemente en un "Staff Meeting" celebrado en el Hospital Universitario Kalorinska, Estocolmo, Suecia, Hospital de Niños que dirige el Profesor Arvid J. Wallgreen, publicado en el "Journal of Pediatrics" del mes pasado, en la que actuaba él como moderador, los Jefes de Servicio de su Clínica expresaron sus ideas a través de sus diversas especialidades. La Dra. Elsa Brita Norlund, psiquiatra infantil manifestaba: que de la revisión de las anamnesis psiquiátrica se desprendía que los niños después de una hospitalización aparecían cambiados en una forma u otra, cambios de di-

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* Conferencia leída en el Hospital de Distrito de Aguadilla, 13 de mayo de 1955, con motivo de la Semana del Hospital.

versa intensidad, relacionando estos cambios con un trastorno del sentido de su seguridad. Muchas madres expresaban, que después del alta, observaban cambios apreciables en la conducta de sus hijos, significando la hospitalización una experiencia trastornadora, debido a que sus hijos no habían llegado a la edad y a la madurez que significaba la separación. Anotando la Dra. Norlund que un Hospital de Niños es un lugar que no resulta innocuo, más cuando al niño cuya edad no le permite expresar una serie de cosas extrañas que ocurren en el Hospital. Y más abajo agrega, que no puede decir a qué edades muestra mayores efectos psicológicos, una separación de la madre; los niños menores de tres años, salidos del Hospital, en el hogar se sienten incómodos y en los mayores de esta edad, en muchos de ellos se observa un abandono total, regresando a una etapa previa del desarrollo, niños que ya habían hablado se tornan silenciosos, aunque no han perdido el don de la palabra, otros que eran alegres se observan taciturnos, presentan enuresis nocturnas, trastornos en el sueño y otros se tornan melancólicos debido a que en forma brusca han perdido la fe en su madre.

Para poder vencer este sentido de inseguridad, ansiedad y temor de parte de los niños, es imprescindible una buena preparación de parte de su madre, antes de su hospitalización, en el cual con persuación, tolerancia y calma se le convence de la necesidad de su hospitalización, asesorada por una enfermera o Trabajadora Médico Social, con el objetivo que no sienta la brusquedad del abandono por parte de ella.

Como pueden apreciar, reviste gran importancia la hospitalización de un niño, en el cual, no solamente hay que considerar el mejoramiento de su trastorno somático, motivo de su ingreso, sino también las alteraciones psíquicas que se pueden derivar de su hospitalización. Debemos tomar en consideración que el psiquis del niño está en plena formación, las nuevas vivencias van a quedar grabadas en su personalidad, y si ellas son notablemente desagradables serán recuerdos difíciles de olvidar en el porvenir, por crear reacciones de miedo y temor que pueden durar a veces muchos años. Así es como podemos explicarnos el terror que significa para muchos niños la presencia de un médico o una enfermera porque ellos los relacionan con exámenes y tratamientos, incómodos, extraños y muchas veces dolorosos. Más todavía si las madres en el hogar estimulan estos hechos.

De observación diaria es ver a muchos niños en las Salas de Pediatría, sumisos y callados unos, inquietos y llorosos otros y ansiosos y aterrorizados también. Estas distintas modalidades de reacción, de acuerdo muchas veces a su temperamento, no es sino la expresión de su inseguridad. Niños que en su mirada buscan

y acogen apoyo en quienes se les acercan y los tratan con dulzura y comprensión.

¿Estamos nosotros en condiciones de atenuar este trauma psíquico que sufre el niño por la separación brusca de su madre? ¿Podremos nosotros contribuir a darles un sentido de seguridad a estos niños que están en un ambiente extraño a su hogar? ¿Contamos con el personal preparado y los recursos necesarios para hacer más llevadera, entretenida y hasta placentera la estadía de estos niños en el hospital? Creemos firmemente que podremos lograrlo, si todos y cada uno de nosotros, nos proponemos a la consecución de este ideal. La atención del niño enfermo, requiere algo más de parte de nosotros, que el frío cumplimiento de nuestras obligaciones: cariño, dulzura, tolerancia y comprensión de estos seres en formación, que debido a sus trastornos somáticos y a sus desarraigos del hogar se tornan aparentemente aún más incomprensibles.

El hospital moderno aunque no sea exclusivamente para niños debe contar con elementos de entretenimiento durante las curas somáticas para prevenir el trastorno mental. La planta física adaptada a un ambiente infantil, con pequeñas unidades o salas, con sus paredes pintadas de colores atractivos y matizadas de figuras propias de su edad; donde se observe una higiene rigurosa y un ambiente tranquilo. Con salas anexas para entretenimiento con muchos y atractivos juguetes y salas o campos de juego para los niños convalecientes.

El "Staff" en todos sus grados desde el empleado hasta el más alto en jerarquía, debe tener un alto sentido de solidaridad y humanismo, tratando de comprender a los niños y a sus madres. Aceptando con dulzura y consideración a los niños, con el objeto que se sientan seguros, liberándolos de su ansiedad por encontrarse en una nueva condición hasta entonces desconocida para ellos; adoptando una aptitud amistosa y comprensiva con sus madres. Cuando se ignoran estos principios, cuando se adopta una aptitud hostíl y malhumorada, cuando se rechaza una madre, peor todavía en presencia de su niño, se desencadena una verdadera crisis, aumenta la inseguridad en el niño, y se crea una desorientación y angustia en la madre. Igualmente fracasa toda terapia cuando se observa una despreocupación general y hasta desdén para los sentimientos y necesidades del niño. Son los médicos y las enfermeras los que más directamente pueden contribuir a darles un sentido de seguridad a los niños. A propósito de ello, la Dra. Norlund dice: que no basta ser médico y de gran preparación si además no hay una sincera comprensión hacia los niños. Y el Dr. T. Ehrenpreis, Cirujano de Niños en el "Staff Meeting" del Hospital Kalorinska decía: Un niño tiene una intuición animal para si una per-

sona le tiene afecto o no, hay que primero hablarle y ganar su confianza. El Pediatra debe ser además un gran psicólogo de niños.

Relaciones con los padres: Ellos deben ser tratados con comprensión, especialmente las madres, que son las que más frecuentemente los traen, ser bienvenidas y aceptadas en el hospital. En sus visitas debe explicárseles la condición clínica de sus hijos. En el Hospital de Clínicas de Buenos Aires, Argentina, las visitas a los niños hospitalizados eran diarias, recibiendo ellas información sobre el estado de sus pacientes. Bien orientadas las madres, las visitas a las salas de Pediatría, no solamente pueden ser útiles como compañía y sosiego de sus hijos, sino además, colaboradoras eficaces en la alimentación y limpieza de ellos, y a veces, de otros niños; elementos de gran ayuda en la orientación de ciertos trastornos y alteraciones del niño. Una condición muy útil, con el objeto de evitar ulteriores trastornos mentales es la presencia de la madre momentos antes y después de las intervenciones quirúrgicas. Otro motivo de inseguridad para los niños y desasosiego para las madres es cuando surge un conflicto entre ella y la enfermera. Obvio está decir que la situación se agrava si el niño es testigo de tal entredicho.

Sería útil y de provecho que las madres pudiesen visitar a sus hijos hospitalizados diariamente, a una hora que no interfiriera las labores en las salas.

Otra forma de atenuar las dificultades de los niños en el hospital, es tratando de acortar en lo posible su estadía.

Estoy seguro que este concepto de Hospitalismo Mental ha fluído constantemente en la mente de todcs nosotros, interpretado de una u otra manera, pero este concepto existe cuando se asila a un niño expresando este Trauma Psíquico de diversa manera e intensidad y su profilaxis es sencilla si todos nosotros ponemos nuestra fe y nuestra decisión, mitigando y haciendo más placentera su estadía, porque en esta forma contribuímos también a la felicidad de nuestros niños.

INFORME A LA CAMARA DE DELEGADOS DE LA ASOCIACION MEDICA DE PUERTO RICO DEL COMITE ESPECIAL DE ESCUDO AZUL

I. Introducción

El día 6 de noviembre de 1954, el Dr. Guillermo Picó, presidente de la Cámara de Delegados de la Asociación Médica de Puerto Rico, nombró un Comité Especial de Escudo Azul, actuando de acuerdo con la siguiente moción adoptada por la Cámara de Delegados:

> "Que por el Presidente se nombre un comité para que formule un plan de Escudo Azul para ofrecer servicios médico-quirúrgicos independientemente del servicio de hospitalización que actualmente ofrece la Cruz Azul de Puerto Rico, y que dicho comité rinda un informe en la próxima reunión de la Cámara de Delegados."

El Comité Especial de Escudo Azul quedó constituído por los siguientes médicos:

Dr. E. Colón Yordán
Dr. Antonio Ramos Oller
Dr. Francisco Raffucci
Dr. Eduardo Pérez Santiago
Dr. Manuel Paniagua
Dr. Víctor J. Montilla

El Dr. Víctor J. Montilla, por considerar que su ideología médica estaba en conflicto con la creación de un plan de esta naturaleza, se excusó de las reuniones del comité y no participó en el estudio que ante ustedes presentamos esta noche.

II. Objetivos y Motivaciones

Es el sentir de los miembros de este comité que la acción de los miembros de la Cámara de Delegados, pidiendo que se realizara este estudio, refleja cierto grado de descontento con los sistemas de seguros de salud vigentes en nuestra isla. Este descontento tiene su origen, entre otros factores, en los siguientes:

1. Que la Cruz Azul de Puerto Rico ofrece servicios a sus suscritores en exceso de lo que razonablemente puede esperarse mediante el pago de primas muy bajas por el suscritor, resultando esto en deterioro en la calidad del servicio médico que

se preste a los pacientes. Muchos de los servicios médicos ofrecidos por el plan son muy costosos y se consideran imposible que puedan prestarse bajo las cuotas actuales.

2. Que estos planes no proveen pagos justos y razonables por servicios médicos especialmente los **no quirúrgicos.**

3. Que en la organización de los planes de seguro de salud operando en la isla los médicos no tienen ningún control inmediato sobre la determinación de lo que deben ser pagos justos y razonables a percibirse por los servicios médico-quirúrgicos que se prestan, habiendo sido muy infrecuentes las revisiones y aumentos de los honorarios profesionales y por el contrario muy frecuentes los aumentos en la cantidad de los servicios médicos a prestarse bajo el plan.

4. Que los médicos consideran que no se protegen los mejores intereses del suscritor al ofrecerle servicios médicos extensos que no puedan ser cubiertos con las asignaciones económicas que ofrecen estos planes a los médicos y hospitales.

5. Que algunos planes se alejan cada vez más y más de las razones primordiales que motivaron su organización, pues en lugar de ofrecer protección para los gastos extraordinarios de enfermedad y accidentes, continúan aumentando la protección para eventualidades triviales que bien pueden ser cubiertas por el presupuesto de la familia corriente, dejando esto poco o ningún margen de protección para aquellas eventualidades costosas, e iniciando un círculo vicioso que necesariamente requiere un aumento en el precio a pagarse por la protección, pero cuyo aumento tampoco se hace, desafiando los más básicos principios actuariales.

6. Que este Plan de Dispensario de la Cruz Azul de Puerto Rico excluye a una gran sección de los miembros de la Clase Médica de participar en el tratamiento de pacientes de la Cruz Azul, que pertenezcan al Plan de Dispensario. Apuntamos inmediatamente que esta situación no prevalece en la ciudad de Ponce en donde el Plan de Dispensario fué modificado por la Asociación Médica del Distrito Sur para poder dar participación igual y equitativa a todos los médicos que así lo deseasen y cuyas credenciales fueran aceptables para prestar servicios a pacientes de la Cruz Azul.

7. Que desde el año 1949 la Cámara de Delegados de la Asociación Médica de Puerto Rico expresó su deseo de no aceptar los planes propuestos por la Cruz Azul, para ofrecer los servicios de dispensario, y nombró un comité para estudiar el problema y a la vez preparar un plan alterno de Escudo Azul en caso de no poderse llegar a un entendido con los representantes de la Cruz Azul de Puerto Rico.

8. Que hasta donde nosotros hemos podido comprobar no ha habido acción posterior de la Cámara de Delegados cambiando su opinión en cuanto a la aceptación de esos planes de servicios médicos de dispensario de la Cruz Azul, por lo cual los médicos entienden que los planes de dispensario no recibieron nunca la aprobación final de la Cámara de Delegados de la Asociación Médica de Puerto Rico.

III. Filosofía Básica de los Planes de Seguro de Enfermedad y Accidentes:

Repetidas veces, la Clase Médica de Puerto Rico, individual y colectivamente, ha expresado su simpatía y respaldo al plan de la Cruz Azul de Puerto Rico. Esto no ha sido otra cosa que la manifestación o exteriorización de nuestra honda inquietud profesional, que nos obliga a velar para que nuestros conciudadanos obtengan la mayor protección médica posible y hacer llegar a todas las personas necesitadas el beneficio máximo de servicios médicos a través de planes voluntarios de servicio de salud.

Personalmente, los miembros de este comité también hemos expresado nuestra conformidad con estos ideales, pero reflexionando más detenidamente hemos creído prudente exponer ante ustedes la filosofía básica que debe regir en todo plan de seguro médico voluntario, así como también el pensar de la Asociación Médica Americana en este respecto antes de respaldar indondicionalmente ningún plan específico de seguro de salud.

Todo plan de seguro de salud voluntario, que incluya servicios médicos, debe estar en armonía con los principios adoptados por la Cámara de Delegados de la Asociación Médica Americana y los cuales anotamos a continuación:

1. **Aprobación Local** — El plan debe tener la aprobación de la Asociación Médica del estado, o si local, de la Asociación Médica de la región o del área en donde el plan opere.

2. **Responsabilidad Profesional** — La profesión médica **debe asumir la responsabilidad** por los servicios médicos incluídos co-

mo beneficios del plan ya que el médico está cualificado, legalmente y por educación, para asumir esta responsabilidad y a aceptar la **responsabilidad por la calidad** de los servicios médicos que se presten.

3. **Libre Elección de Médico** — No puede haber ninguna restricción a la libre elección de un médico, debidamente cualificado y autorizado a practicar la medicina en la localidad en que opere el plan, siempre que éste esté dispuesto a rendir servicios bajo las condiciones establecidas por el plan.

4. El plan debe conservar y respetar la relación de paciente y médico.

5. Todo plan que requiera por escrito que los médicos participantes se abstengan de participar en alguno otro plan similar, será considerado inelegible para recibir el sello de aceptación de la Asociación Médica Americana.

IV. **Origen de los Planes**

Los planes voluntarios de seguro de salud que ofrecen una libre elección de médico tienen un origen reciente. El interés en estos planes fué motivado probablemente por la depresión del año 1929, habiendo tomado gran impulso el movimiento de organización durante los primeros años de la década de 1930 al 1940.

Los primeros planes voluntarios de seguro médico y de hospitalización se desarrollaron extensamente en el Oeste de los Estados Unidos, especialmente en los estados de Washington y Oregon. Fué durante el período del 1930 al 1940 que los planes de beneficios de hospitalización fueron organizados, recibiendo el respaldo de hospitales, asociaciones de hospitales y sociedades médicas.

Desde el comienzo de estos planes, la Cámara de Delegados de la Asociación Médica Americana formuló ciertos principios para ser observados por estos planes y uno de estos principios establecía que los contratos por **servicios de hospitalización** no deberían incluir servicios médicos. En el 1937, en la reunión de la Cámara de Delegados, se adoptó la siguiente resolución:

> "Si por alguna razón existe la necesidad de incluir servicios médicos especiales como Radiología, Patología, Anestesia y servicios médicos de dispensario para pacientes ambulatorios, estos servicios podrán ser incluídos en los planes sólo bajo la condición de que se hagan pagos de dinero en efec-

tivo por la organización directamente al suscritor por los costos de estos servicios."

Años más tarde, la Cámara de Delegados de la Asociación Médica Americana volvió a expresarse de la siguiente manera:

"1. Todos los aspectos de servicios médicos en cualquier metodología de práctica médica deberán estar **bajo el control de la profesión médica.** Ningún otro cuerpo o grupo de individuos está legalmente y educacionalmente equipado para ejercitar ese control.

2. A ninguna tercera persona se le permitirá intervenir en las relaciones médicas entre el paciente y su médico.

3. El paciente tendrá absoluta libertad para elegir su médico, si éste está legalmente cualificado para ejercer la medicina o si está dispuesto a prestar el servicio.

4. Los aspectos médicos de todas las instituciones que están envueltas en el proceso de rendir servicios médicos deben estar bajo control profesional, entendiéndose que servicios de hospital y servicios médicos deben ser considerados separadamente. Estas instituciones son simplemente extensiones del equipo del médico. El médico es el único a quien las leyes de todas las naciones reconocen como competente para usarlas al rendir servicios médicos. La profesión médica solamente puede determinar la bondad y carácter de esas instituciones y su valor depende de que sean operadas de acuerdo con niveles médicos adecuados."

Durante los años iniciales del movimiento de organización de planes de seguro de salud no se hicieron grandes esfuerzos para desarrollar planes de servicios médicos, lo cual se debió a:

1. La falta de experiencia y estudios actuariales.

2. La vacilación de los médicos en aventurarse en un proyecto de esta naturaleza sin haber tenido el beneficio de experiencia previa.

3. La falta de orientación del público en cuanto a las bondades y beneficios que estos planes pueden ofrecer.

Originalmente, la Asociación Médica Americana, en el año 1938, aconsejó que solamente beneficios en efectivo fueran paga-

dos a los suscritores por planes de seguro de salud ya que esto constituía el método más efectivo para no alterar las relaciones entre pacientes, médicos y hospitales; pero en el año 1942 la Cámara de Delegados reconsideró su resolución anterior y aprobó una nueva resolución mediante la cual aprobaba el principio de que planes de seguros de salud ofrecieran como beneficio **servicios médicos**, si estaban respaldados por una sociedad médica del estado o del territorio en el cual operara el plan.

En los Estados Unidos existían hasta el año 1953, 82 planes voluntarios de seguro de salud, respaldados por asociaciones médicas incluyendo este número aquellos planes que trabajan en coordinación con la Cruz Azul. El número total de suscritores de estos planes es de 31-1 3 de millones de personas de un total de casi 100 millones que están cubiertas de una manera o de otra por planes voluntarios de seguro de salud.

En el año 1954, el número de planes auspiciados por sociedades médicas en los Estados Unidos ya había aumentado a 103. Hay en actividad 79 planes de hospitalización de tipo Cruz Azul, incluyendo este número los del Distrito de Columbia y Puerto Rico, y más de 500 compañías de seguro de carácter privado, ofreciendo simultáneamente a grupos e individuos seguro médico y de hospitalización. Además de estas 500 compañías privadas de seguro hay en funcionamiento alrededor de 100 planes independientes que son auspiciados por cooperativas rurales, industrias, empleados y grupos de diferentes uniones.

Todos reconocemos y aceptamos que desde el punto de vista del individuo los costos de enfermedades y accidentes son siempre inciertos, impredecibles y generalmente inoportunos. El peso de estos gastos inesperados generalmente puede ser aliviado por una acción coordinada de grupo mediante la cual muchas familias e individuos contribuyen con dinero a un fondo común de donde pueda pagarse los servicios de hospitalización y servicios médicos, cuando la situación así lo requiera. De esta manera el factor de la incertidumbre se elimina y se reemplaza o substituye por la certeza de un gasto fijo y estable, lo cual es la prima que se paga por el seguro de salud.

Esto constituye la base en que descansan los principios de seguros voluntarios de salud y prepara al paciente para poder afrontar gastos imprevistos por enfermedad o accidentes aunque tengamos que admitir que los planes actuales no proveen todavía suficientemente para cubrir las verdaderas enfermedades y accidentes de carácter catastrófico, durante las cuales el paciente necesita la mayor ayuda por estar imposibilitado de los medios para proveer para él y su familia durante el período de enfermedad; además de los gastos que la enfermedad de por si conlleva. La

mayor parte de los planes cubren gastos de hospitalización y servicios médicos para enfermedades cortas con limitaciones de tiempo de hospitalización, etc., lo cual no resuelve para el suscritor el problema de las enfermedades catastróficas.

La Asociación Médica Americana ha dado su definición de seguro médico como "un método de transferir el peso económico que acarrea las enfermedades del individuo al grupo."

Todo médico y paciente debe tener siempre presente en su mente el verdadero concepto de seguro de salud voluntario que abraza la filosofía básica de que estos mecanismos son sólo una ayuda para el recipiente de los servicios médicos y de hospitalización en el financiamiento de estos servicios y "por lo tanto es claro que la filosofía básica de estos planes se funda en el claro concepto de que al obtener seguros de salud el paciente no está comprando una póliza que cubre totalmente los gastos en que va a incurrir sino que más bien tendrá una sustancial ayuda para el pago de los servicios de hospitalización y servicios médicos.

No existe armonía total entre las diferentes autoridades en cuanto si el costo total incidental a la prestación de servicios médicos y hospitalización son sujetos apropiados para asegurarse. Si algunos costos en el cuido de la salud son sujetos favorables a los principios de seguro, la mayor parte de las autoridades opinan que exámenes físicos de rutina, exámenes rutinarios, visitas a la oficina del médico y visitas a la casa por el médico, para enfermedades de corta duración deben ser incluídas en el presupuesto de la familia y no estar incluídas como parte del seguro contra enfermedad.

De acuerdo con los principios de seguro, no se pueden incluir factores de pérdidas, tales como los arriba mencionados, los cuales son bien conocidos y de una incidencia conocida; dentro de un programa de seguros es necesario aumentar el importe de la prima para estas contingencias además de la prima calculada para las pérdidas imprevistas y si a esto le añadimos el cargo de administración, llegamos a la conclusión de que finalmente el suscritor estaría pagando mucho más por servicios médicos de rutina que si no tuvieran seguro médico.

Por consiguiente, debemos aceptar el principio de que el seguro de salud voluntario está primordialmente dirigido u organizado para aquellas personas que pueden normalmente, dentro de sus presupuestos, hacerse cargo de los gastos usuales que conllevan enfermedades de rutina, pero para los cuales una seria enfermedad significaría una gran catástrofe financiera.

Los planes de seguro de salud han recibido el más entusiasta endoso de la Clase Médica en los Estados Unidos, pero para poder continuar creciendo y mejorando los servicios que ofrecen al pue-

blo requieren el más íntimo entendimiento y cooperación de los suscritores, de los hospitales y de la profesión médica.

El pueblo americano ha desarrollado y logrado su independencia económica y sus grandes adelantos médicos a base de la iniciativa propia y la actuación voluntaria del individuo. El progreso de los Estados Unidos ha sido impulsado por la actuación voluntaria del individuo y es solamente en esta base que probablemente se pueda asegurar la total solución del problema de servicios médicos en años futuros.

Reconocemos que hay una infinidad de problemas que resolver aún en lo que concierne a los planes de seguro de salud voluntarios. Si la mayoría de los pacientes que se acogen a los beneficios de estos planes de seguro insisten en continuar creyendo que estos planes deben ofrecer garantía para cubrir los gastos médicos en su totalidad, y si también los dueños de hospitales o las juntas directivas de los hospitales y los médicos individualmente asumen este punto de vista, el resultado no puede ser otro que un obstáculo que impedirá a estos planes llegar al desarrollo máximo de su efectividad comunal.

Otro de los problemas ya mencionados y de gran importancia es la clasificación impropia de los servicios profesionales dentro de los servicios institucionales por algunas de las organizaciones aseguradoras. Repetidas veces, diferentes autoridades han definido y hecho claro la diferenciación entre servicios médicos y servicios institucionales, pero la continua y constante ignorancia y desprecio a estos principios, postulados por la Asociación Médica Americana, ha hecho que en diferentes ocasiones la Cámara de Delegados de la Asociación Médica Americana reitere sus acciones y pronunciamientos previos.

En la Sesión Clínica de St. Louis, en el año 1939, la Cámara de Delegados de la Asociación Médica Americana aprobó una resolución que en parte decía:

> "El contrato del suscritor debe excluir todo servicio médico — las provisiones del contrato deben ser exclusivamente limitadas a facilidades del hospital."
>
> "Si el servicio de hospital se limita a incluir solamente el cuarto en el hospital, cama, comida, sala de operaciones, medicinas, vendajes y materiales quirúrgicos y cuido de enfermería en general, la distinción entre servicios de hospitalización y servicios médicos estará clara."

Los planes de Cruz Azul son auspiciados por los hospitales y aprobados por la Comisión de Cruz Azul de la Sociedad Americana de Hospitales. La aprobación de los planes de la Cruz Azul se basa

en que se llenen ciertos requisitos en cuanto a la representación de hospitales, médicos y los suscritores, que sea con fines no pecuniarios, que tenga reservas adecuadas, que ofrezca libre elección de médico y hospital, que tenga estudios actuariales adecuados, etc. Estos planes son generalmente supervisados por el Superintendente de Seguro del estado.

Con esta orientación en cuanto a la filosofía básica de los planes, podemos entrar en una breve exposición de los beneficios que proveen los planes de seguro de salud.

Generalmene hablando, los planes que se organizan para prestar servicios médicos se pueden clasificar de tres maneras, de acuerdo con los beneficios que proveen. Los tres tipos de beneficios son los siguientes:

1. Indemnidad;
2. Servicio;
3. Combinación de Indemnidad y Servicio

Existen múltiples variaciones y adaptaciones de estos tres tipos básicos de beneficios.

En el plan de **Indemnidad** se provee beneficios en término de dinero, establecido en tablas de acuerdo con las enfermedades, días de hospitalización, etc. Estos beneficios en efectivo se aplican como créditos hacia el costo total del servicio profesional que el paciente ha recibido.

El plan de **Servicio** es aquel en el que al suscritor se le promete y recibe beneficios en forma de servicios quirúrgicos o médicos sin costo adicional para el suscritor.

Cuando se combinan los dos tipos de servicio arriba descritos, o sea Indemnidad y Servicio, tenemos el tercer plan que es el que se denomina **Combinación.** En este tipo de plan, a suscritores que tengan entradas anuales bajo cierto límite establecido de antemano, y que se acomoden a ciertas facilidades de hospital, establecidas en el contrato, se les promete servicios médico-quirúrgicos generalmente sin cargo adicional. En caso de suscritores cuyas entradas o ingresos anuales excedan la cantidad especificada, o aquellos que demandan o piden facilidades de hospital de más precio que las especificadas en el contrato, los beneficios que recibe en la tabla de Indemnización se acreditan al paciente hacia el costo total del servicio médico rendido.

Estos tres tipos de beneficios son generalmente usados por las cuatro organizaciones o agencias que se dedican a la venta de seguros de salud, o sea los planes de las sociedades médicas, los planes de la Cruz Azul, las compañías de seguro privado y los planes de empleados y otros grupos.

Los planes de seguro médico voluntario, ofrecidos por la profesión médica, han adquirido en los últimos años una importancia inestimable en el panorama de los servicios médicos y sin duda alguna en el curso de los próximos años tendrán aún más influencia y peso en el futuro inmediato de los principios democráticos, y no hay duda que afectarán grandemente la práctica futura de la medicina en Puerto Rico.

El desarrollo de estos planes ha sido de tal magnitud que ha sido clasificado como uno de los más grandes desarrollos de esta naturaleza en la historia, ya que ni en el campo de la religión, ni de la política, ni en ningún otro campo sociológico se han podido agrupar de 85 a 100 millones de personas para su propio beneficio en un tan corto período de tiempo.

El Dr. Louis Bauer, ex-presidente de la Asociación Médica Americana, expresó su opinión recientemente de que eventualmente más de un millón de personas serían cubiertas por los planes de seguro médico voluntario. Este número sería, en su opinión, suficiente ya que alrededor de 25 millones de personas reciben servicios médicos en parte o en totalidad del Gobierno Americano; 10 millones no creen en servicios médicos, prefiriendo comprar lo que necesitan en las farmacias o reciben consejos de cultistas; cinco millones son indigentes, cuyo servicio médico está en manos de organizaciones locales; y cerca de 10 millones no están interesados en ningún aspecto de seguro de salud voluntario.

La opinión de autoridades competentes en este asunto es que una de las virtudes principales de los planes de seguro voluntario de salud es que no pagan el costo total de cada renglón del servicio médico. Una protección total representaría probablemente una violación a los más sólidos principios actuariales y elevaría grandemente los costos administrativos. Se aduce también que pólizas de protección total constituyen la esencia de la medicina gubernamental y es lo que tiende a poner en bancarrota estos planes gubernamentales.

Debido al gran número de personas que opinan que los planes de seguros médicos son ineficientes ya que no proveen protección contra las enfermedades de carácter catastrófico, las cuales generalmente destrozan los presupuestos de las familias y sin duda alguna también proveen las mejores razones a aquellos que propulsan planes de servicio médico compulsorios bajo control gubernamental, ya hay varias entidades que están empezando experimentos en el campo del seguro para enfermedades catasfróficas.

Entre los primeros en este experimento está el California Physician Service, quienes por una pequeña prima adicional ofrecen a sus suscritores protección total por dos años contra el costo

de 23 de las temibles enfermedades, entre ellas Cáncer, Tuberculosis y Poliomielitis.

No es de dudarse que dentro de los próximos años la gran mayoría del pueblo americano esté cubierto de una manera o de otra por planes de seguro de salud voluntario, pero para esto se necesita romper las barreras que ha impuesto hasta ahora la apatía de la gente hacia la protección médica que deben tener y cuyos beneficios no aprecian hasta el momento en que debido a una enfermedad tienen que hacer erogaciones fuera del alcance de sus medios, siendo tarde ya para quedar protegidos por ninguno de estos planes; y además la apatía de los médicos al no estudiar el problema de los seguros de salud voluntarios para poder estimular y aconsejar honradamente a sus pacientes para que se provean de esta protección contra los inesperados e inoportunos gastos que enfermedades y accidentes conllevan.

Debido a la competencia normal entre las compañías de seguro privadas, planes independientes y Escudo Azul, el paciente ha ganado grandemente ya que todos los competidores en este campo deben estar de acuerdo en que el factor importante es que el suscritor tenga protección adecuada, sin importar el tipo de agencia que la provea.

Solamente bajo un sistema competitivo, con provisiones para la libre elección del plan que el paciente desea, podrá llegarse a cristalizar el plan ideal de seguro de salud voluntario ofrecido al más bajo costo y rindiendo los más satisfactorios beneficios médicos.

Los planes de Escudo Azul, para poder llegar a aportar el máximo servicio al público, tienen que recibir la aprobación y el respaldo incondicional de la profesión médica, quien debe ver estos planes de seguro médico como planes de su propia creación. Estos planes son hijos de la medicina organizada. No son una compañía de seguro, sino el mecanismo eficiente y efectivo creado, por el propio médico, para hacer llegar los mejores servicios médicos al mayor número de personas.

V. Situación Actual en Puerto Rico

En nuestra isla funcionan, en el campo de seguro de salud voluntario, los siguientes planes:

1. Veinte compañías de seguro independientes, autorizadas para hacer seguros de enfermedad y accidente, y debidamente registradas en la Oficina del Superintendente de Seguro.

2. Varios planes independientes, controlados por médicos individualmente o grupos de médicos.
3. Planes independientes de uniones de trabajadores, empleados, maestros, etc.
4. La Cruz Azul, la cual vende seguros de hospitalización y de servicios médico-quirúrgicos.

A. Planes Independientes

Las veinte compañías de seguro comercial, que operan en Puerto Rico bajo la supervisión del Superintendente de Seguro de Puerto Rico y al amparo de la Ley General de Seguros, cobraron durante el año 1954 primas ascendentes a $498,696.11 y pagaron pérdidas durante el mismo período montante a $257,277.77.

Estas compañías independientes de seguro ofrecen generalmente beneficios de indemnidad, pagando al paciente una cantidad estipulada para reembolsarle por los gastos de hospitalización y servicios médicos, aunque algunas tienen contratos independientes con los hospitales y pagan directamente al hospital por los gastos incurridos por el suscritor.

Hay otro grupo de planes independientes que operan bajo la dirección de médicos con médicos a sueldo. No es necesario mencionar que algunos de estos tipos de arreglo puede objetarse éticamente ya que probablemente este método de práctica médica envuelve la práctica contractual de la medicina, limitando o restringiendo la libre elección de médico por el paciente y hasta cierto punto interfiriendo con la libre competencia entre los médicos.

La Corte Suprema de los Estados Unidos, en su opinión emitida en el caso de los Estados Unidos de América contra el Oregon State Medical Society, estuvo en completo acuerdo con las objeciones de los médicos del estado de Oregon a formas de práctica contractual de la medicina en la cual un patrono o compañía de seguro se constituye en una tercera persona en las relaciones entre médico y paciente, y resuelve que si el médico depende del patrono o de la compañía de seguro para el pago de sus honorarios profesionales, en lugar de depender del paciente, el médico está sirviendo a dos amos con intereses conflictivos.

La práctica contractual de la medicina constituye, al efecto, un arreglo o convenio entre un médico y un grupo de médicos como principales o agentes de una compañía, organización o individuo, para rendir servicios médicos totales o parciales a un grupo o clase de individuos, basándose en un sueldo o un pago estipulado por cada paciente examinado o tratado. La práctica contractual en sí no constituye necesariamente una violación a la ética médica. Se

constituye en práctica no-ética si existen ciertos aspectos tales como:

1. Si solicitan pacientes directa o indirectamente.
2. Cuando interfiere con la competencia razonable que debe existir en la comunidad.
3. Cuando la libre elección de médico no puede ser ejercida por el paciente.
4. La libre elección de médico, cuando se aplica el término en relación a la práctica contractual, es definida por la Asociación Médica Americana como "ese grado de libertad en la selección del médico que se puede ejercer bajo las condiciones usuales de empleo entre médico y paciente," cuando ninguna otra persona tenga "interés válido" o intervenga.

La existencia de una tercera persona con intereses válidos no hace que un contrato sea necesariamente no ético. El "interés válido" se define como ese interés que por ley o necesidad hace que una tercera persona sea legalmente responsable por el costo del servicio médico o por la indemnidad. Es obvio que un plan de seguro de salud carece del "interés válido", según se define en el Código de Etica, cuando es controlado u operado por personas que no sean médicos incluyendo los consumidores o recipientes del seguro médico.

Durante el curso de este estudio, hemos dedicado algún tiempo a compenetrarnos, hasta donde nos ha sido posible, con la organización y operaciones de la Cruz Azul de Puerto Rico ya que esta entidad ha sido la organización primera en ofrecer servicios de hospitalización y servicios médico-quirúrgicos bajo un plan de seguro de salud voluntario en la isla y actualmente cuenta con un gran número de suscritores.

Queremos repetir que somos firmes creyentes en el principio de los planes de seguro de salud voluntario, a través de organizaciones como el Escudo Azul y otros planes similares, que puedan ofrecer a nuestro pueblo los medios para enfrentarse a la siempre presente amenaza de gastos inesperados de hospitalización y servicios médicos que puedan crear caos económicos y torturas mentales en las familias que por falta de recursos económicos no pueden sufragar estos gastos; pero también queremos hacer claro que estamos en completo desacuerdo con todos los planes que, aunque organizados con este noble propósito en mente, violan los principios básicos de las relaciones profesionales y cuyas actividades al ser aceptadas tácita o abiertamente por la Clase Médica puedan afectar desfavorablemente el desarrollo futuro de otros planes similares, alterando y rebajando la calidad de los servicios médicos y fomentando aún más el desprecio por las ya en muchas

ocasiones ignoradas y olvidadas normas y principios de la Etica y Moral Médica.

De no enfrentarnos nosotros al problema ahora, vemos en el horizonte el no lejano momento en que estas prácticas, aunque no motivadas por deseos mezquinos y si más bien el resultado de una ignorancia completa de los postulados éticos más elevados que nos deben regir, puedan constituirse por costumbre en el ejemplo que adopten otras agrupaciones al organizarse en años futuros. Es necesario cortar el mal de raíz ahora, antes que tome tal magnitud que se acostumbren los médicos y otras personas envueltas a creer que es el método correcto de hacer las cosas.

No dudamos que en el caso específico de la Cruz Azul las irregularidades, que en nuestra opinión existen y las cuales pasaremos a anotar inmediatamente, no han sido motivadas por el deseo de violar nuestros principios sino más bien han sido el resultado de las dificultades que ha tenido este plan en sobrevivir desde su comienzo y el deseo honrado de extender los beneficios de un plan como la Cruz Azul al mayor número de personas en la isla. Con estas observaciones en mente vamos a pasar a hacer un breve análisis de algunos de los puntos que consideramos más importantes dentro de la organización de la Cruz Azul de Puerto Rico.

La Cruz Azul de Puerto Rico fué organizada al amparo de la Ley #152, aprobada en el mes de mayo de 1942, y cuyo propósito es "autorizar la formación de asociaciones con fines no pecunarios para **prestación de servicios de hospitalización** bajo la supervisión del Superintendente de Seguros de Puerto Rico y para otros fines."

Dice la ley, en su texto, que las organizaciones creadas al amparo de esta ley estarán exentas del cumplimiento de las disposiciones de la Ley de Seguro de Puerto Rico con excepción de las que en ésta se provea específicamente.

Haciendo un estudio de la ley básica #152, encontramos que dice: "los contratos entre la solicitante y los hospitales solicitantes **obliga al hospital o grupos de hospitales,** que sean parte en el contrato, a prestar los servicios a cada suscritor a que tuviera este derecho bajo los términos del contrato que se le hubiera extendido.

El día 25 de abril de 1946, la Ley #466 fué aprobada para proveer **servicios médicos y de hospitalización** a los funcionarios y empleados del Gobierno Insular de Puerto Rico. El artículo V, Inciso B, autoriza al Superintendente de Seguro de Puerto Rico a **"formalizar con la Cruz Azul el contrato o los contratos necesarios para la prestación de los servicios médicos y de hospitalización"** a que se refiere esta ley.

El 31 de diciembre de 1946 se aprobó la Ley #21 para adicio-

nar dos incisos que se denominaron Inciso A e Inciso B de la Sección I de la Ley #152 del 9 de marzo de 1946.

El Inciso A. en parte dice: "Por **atención de hospital** o **servicios de hospitalización,** tal y como se expresa en la sección anterior y a los efectos de esta ley, se entenderá todos los servicios que pueda prestar un hospital en **cualquiera de sus departamentos** incluyendo en estos el de dispensario y servicio social así como cualquier otro necesario para la conservación de la salud o el completo restablecimiento del paciente."

El Inciso B en parte dice: "Hasta donde sea posible, todo servicio que se preste bajo el plan que especifique esta ley **deberá ser uniforme en cuanto a los servicios que deberán prestarse a los suscritores** y este requisito deberá conseguirse en todo contrato que se formalice con hospitales o clínicas previamente clasificados."

La Ley #196 del 7 de marzo de 1949 y en su sección III dice en parte:

> "La Junta de Directores de una asociación organizada con fines no pecuniarios para establecer, mantener y operar un plan de servicios de hospitalización estará formada por grupos de las siguientes personas:
>
> 1. Administradores, directores o síndicos de hospitales que hubieran contratado con dicha asociación para rendir servicios de hospitalización a sus suscritores.
> 2. **Doctores en el ejercicio de la Medicina y Cirugía,** que no estén incluídos en el grupo #1 y en número igual al número que compongan el grupo #1.
> 3. Personas del público, que fueran suscritores de dichos servicios de hospitalización, en un número igual a la suma de los grupos #1 y #2 arriba mencionados."

La Ley #311 del 13 de mayo de 1949, aprobada con el propósito de enmendar el título y los artículos II y III de la Ley #462 del 25 de abril de 1946, autoriza a las Asambleas Municipales de Puerto Rico incluyendo la Junta de Comisionados del Gobierno de la Capital, a proveer, mediante ordenanza, **servicios médicos y de hospitalización** bajo un plan análogo al que se estableció en esta ley para los **funcionarios y los empleados de los gobiernos municipales.**

Las leyes arriba mencionadas autorizan la organización, con fines no pecuniarios, de agencias para prestar **servicios de hospitalización.** En la ley original, o sea la Ley #152, no se hace mención ninguna de **servicios médicos** y esta ley **obliga al hospital** a prestar los servicios al suscritor a que tengan derecho bajo el contrato **expedido.**

Es la Ley #466 la que hace mención por primera vez de **servicios médicos,** pero autoriza la prestación de estos servicios, o sea **servicios médicos y de hospitalización,** sólo a los empleados y funcionarios del Gobierno Insular; más tarde se autoriza a las Asambleas Municipales y Junta de Comisionados del Gobierno de la Capital, por virtud de la Ley #311, a proveer **servicios médicos y de hospitalización** bajo un plan análogo a los funcionarios y empleados de los gobiernos municipales.

Entendemos pues que la ley solamente autoriza a la Cruz Azul a prestar **servicios médicos** a estos dos grupos de empleados de los gobiernos insulares y municipales. En la definición de **servicio de hospitalización,** que se hace en el Inciso A de la Ley #466, se autoriza a los hospitales a prestar servicio de dispensario. No creemos que esto pueda interpretarse como que deba incluir los servicios profesionales, o sea los servicios médicos prestados en los dispensarios, y si más bien que se refiere a las facilidades físicas de dispensario como servicios de enfermeras, archivos, tarjeteros y otros requisitos que son necesarios para el establecimiento de un dispensario.

Al decir la ley que por **servicios de hospitalización** se entenderá todos los servicios que **puede** prestar un hospital en sus diferentes departamentos, no puede entenderse que esto incluye servicios profesionales de los médicos ya que la Facultad Médica no forma parte de ningún departamento del hospital, sino es una organización con gobierno propio y en quienes la Junta de Directores del hospital delega totalmente la responsabilidad de ejercer todas las funciones médicas dentro del hospital.

Hemos estudiado la comunicación de la Oficina del Secretario de Justicia, suscrita por el Secretario de Justicia, Hon. José Trías Monje, que con fecha del 26 de abril de 1954 dirigiera al Superintendente de Seguros de Puerto Rico, Sr. M. Nieves Hidalgo. Esta carta del Procurador la motivó una pregunta hecha por el Sr. Nieves Hidalgo sobre si podía organizarse en Puerto Rico una entidad para prestar servicios médicos **exclusivamente** sin incluir servicios de hospitalización. La opinión del Procurador General es que no puede organizarse una asociación para prestar servicios médicos exclusivamente sin incluir el servicio de hospitalización bajo las disposiciones de la Ley #152 del 9 de mayo de 1942. Debemos mencionar que una opinión previa de la Oficina del Procurador, con fecha del 16 de agosto de 1946, definiendo el sentido y el alcance de la frase "servicio de hospitalización", dice en parte: ". . . . incluye únicamente aquellos **servicios médicos** que deben prestarse a un paciente hospitalizado". Para poder entonces extender el servicio a pacientes no hospitalizados fué necesario

enmendar la ley, lo cual se hizo a través de la Ley #21, que ya mencionamos.

El propio Procurador General, en su carta del 26 de abril de 1954, dice:

> "En Puerto Rico hay dos estatutos sobre la materia, la Ley #152 del 9 de mayo de 1942 y la Ley #466 del 25 de abril de 1946. La primera autoriza la formación de asociaciones con fines no pecuniarios para establecer, mantener y operar cualquier plan de **servicio de hospitalización** sin fines pecuniarios, y en virtud del cual se provea **atención de hospital por algún hospital o grupo de hospitales.**"

Consideramos que la opinión del Procurador no puede interpretarse como que en Puerto Rico los hospitales están autorizados para prestar servicios médicos. Esto simplemente sería la práctica corporativa de la medicina. Hay suficientes opiniones y decisiones legales que han respaldado el principio de que no puede practicarse medicina por corporaciones, y todas estas opiniones han surgido en relación a casos en que las corporaciones han sido creadas con fines pecuniarios por médicos o personal no médico.

Si el hospital hace un contrato para prestar servicios médicos, el hospital está practicando medicina a través de sus agentes, los médicos, y de acuerdo a la doctrina legal los actos del agente son los actos del superior. Ninguna persona que no sea un médico autorizado puede directa o indirectamente practicar medicina. Solamente personas naturales pueden ser licenciadas para practicar medicina. Las corporaciones no tienen derechos profesionales, privilegios o poderes. Es una imposibilidad física que una corporación pueda practicar medicina ya que no pueden ceñirse a los estatutos tomando exámenes, pasando exámenes, etc.

Estamos seguros que esa opinión del Procurador General hizo que la Cruz Azul de Puerto Rico extendiera su campo de actividades para vender servicios profesionales a los suscritores, además del servicio de hospitalización.

Si el servicio de hospitalización fuese el único que la Cruz Azul ofreciera en sus contratos, solamente estaría en conflicto la paternidad de las especialidades de Patología, Radiología, Anestesia y Terapia Física, las cuales son incluídas como servicios de hospitalización en algunos planes, incluyendo los de la Cruz Azul, y como servicios médicos en otros y los cuales la Asociación Méca Americana opina enfáticamente que no deben incluirse con los servivcios de hospitalización o en planes de hospitalización.

La Cruz Azul, al asumir que la ley le autoriza para la extensión de sus actividades al campo médico-quirúrgico, ha entrado en este aspecto de la práctica médica haciendo contratos con médi-

cos a través de hospitales e individualmente. Siendo esto cierto, entonces señalamos nuestra objeción ética de que la Cruz Azul no tiene el interés válido, según la definición que dimos anteriormente y que ha sido postulada por la Asociación Médica Americana, y se constituye en una tercera parte o una tercera persona en las relaciones entre médico y paciente. Además los médicos no tienen control ninguno sobre los servicios médicos; y varios aspectos de este plan están en contra de los principios de la libre elección de médico, a saber el Plan de Dispensario.

La profesión médica no tiene control absoluto sobre la ética de ninguna persona excepto sus propios miembros, no tiene control de ninguna naturaleza sobre agencias de seguro ya sean compañías comerciales o compañías voluntarias, con fines no pecuniarios, a quienes el médico le da el poder de vender sus servicios. Esta es la razón por la cual no puede existir ninguna base ética bajo la cual un médico pueda permitir a una agencia lega a vender sus servicios por un precio sin esto constituirse en una condición que tiende a la explotación del paciente.

El Plan de Dispensario de la Cruz Azul de Puerto Rico es un ejemplo más de una situación irregular. En el área metropolitana existen aproximadamente, según la información que hemos podido obtener, 13 dispensarios de la Cruz Azul siendo la mayor parte de ellos dispensarios de hospitales y algunos dispensarios de médicos individuales. Este tipo de plan limita la participación de todos los médicos que estén dispuestos a servir a pacientes de la Cruz Azul y si bien puede alegarse que cualquier médico puede participar del Plan de Dispensario en el área metropolitana, que tenga facilidades de radiología, laboratorio, etc., exigidos por el plan, es lógico asumir que la práctica médica corriente no exige al médico tener este equipo necesariamente para prestar servicios médicos adecuados en su oficina, por lo cual los requisitos de la Cruz Azul están limitando indirectamente la libre elección del médico por el paciente.

Aún asumiendo que la ley autoriza a los hospitales a practicar medicina, la enmienda provista por la Ley #21 autoriza solamente a los **hospitales a proveer servicio de dispensario.** No hemos encontrado en ninguna parte de la ley nada que indique que los médicos individualmente en su oficina puedan prestar servicios de dispensario bajo contrato con la Cruz Azul.

El Sr. Mark Kormes, actuario consultor de la Cruz Azul de Puerto Rico, expresó su opinión de que la asignación de pacientes a hospitales estaba en conflicto con la libre elección de médico por el paciente, y aconsejó que esto no debía hacerse; pero encontramos que los pacientes acogidos al Plan de Dispensario están actualmente asignados a diferentes dispensarios y, como antes deja-

mos apuntado, la Junta de Regentes tiene el control de la aceptación o rechazo de solicitudes de cambios de dispensario.

No es necesario discutir más el problema ya que para nosotros es obvio que esto está en contra de las normas básicas de la práctica médica, según los postulados de la Asociación Médica Americana. El Sr. Kormes, en su carta del 19 de octubre de 1953, dirigida a la Cruz Azul de Puerto Rico, también expresó muy bien el hecho de que la idea de la Cruz Azul de establecer sus propios dispensarios sería rechazada por la profesión médica y añadió la frase "and properly so".

En varias ocasiones la Cruz Azul de Puerto Rico ha formulado planes encaminados a establecer dispensarios propios; y en su carta del 23 de julio de 1953 al Comité Ejecutivo, el Director Ejecutivo esbozó un plan mediante el cual la Cruz Azul establecería sus propios dispensarios médicos, brindando servicios médicos a través de médicos asalariados. Si bien estos esfuerzos por establecer los dispensarios con médicos asalariados, pagados directamente por la organización de la Cruz Azul, aparentemente no han tenido el respaldo de la Junta de Regentes, ha habido repetidas intenciones de establecer este tipo de servicio. Un plan similar a este, mencionado en la comunicación anterior, trató de establecerse en la ciudad de Ponce para dar servicio a una unión de trabajadores de muelles, no habiendo recibido el endoso de la Asociación Médica del Distrito Sur. La importancia que damos a esto se basa en que vemos una constante tendencia a establecer métodos de práctica médica que están completamente en desacuerdo con nuestros postulados.

Si uno de estos planes de dispensario hubiese sido aceptado y estuviese en funcionamiento, veríamos en ellos el principio de un cambio radical en nuestros sistemas médicos ya que a menos que hubiese sido objetado por los miembros de la Asociación Médica de Puerto Rico, el aumentar en número estos dispensarios, traerían una competencia ilícita y perjudicial para los médicos que practicaran en la comunidad donde estos dispensarios fueran establecidos.

¿Por qué se pensó en el establecimiento de estos dispensarios? No lo sabemos y no creemos que el propósito primordial haya sido el violentar e ignorar el sentir de la profesión organizada sino más bien, como dijimos anteriormente, la necesidad de hacer algo para extender servicios médicos a un mayor número de personas. Por eso es que **debe ser la Asociación Médica de Puerto Rico** la que tome esta iniciativa y la tome con el conocimiento y convencimiento absoluto de que el método a seguirse ha de ser el método correcto y que no se implantarán costumbres o métodos

que sean perjudiciales al desarrollo normal de la práctica médica en la isla.

Nos ha sorprendido grandemente el estudio que hemos hecho de los contratos que tiene la Cruz Azul con los hospitales de Puerto Rico. El contrato típico de esta organización con un hospital dice en su primera página:

> "La Cruz Azul de Puerto Rico y el hospital **X**, corporación que se dedica a la **hospitalización y tratamiento médico-quirúrgico en general...**"

La primera frase del contrato expresa una ignorancia total de la función del hospital y lo constituye en un practicante de la medicina. Más adelante dice el contrato:

> "Las partes comparecientes tienen convenido un contrato para la hospitalización y asistencia médica-quirúrgica en general y lo llevan a efecto bajo las siguientes cláusulas."

Esto es una repetición del error anterior. La Cláusula I, Inciso E del mismo contrato dice:

> "El término servicio de hospitalización significará todos los servicios especificados en la Cláusula III de este contrato."

Y la Cláusula III dice en su Sección B:

> "Servicios Médico-Quirúrgicos — El Hospital X se compromete y obliga a que su **Staff Médico**, según esté registrado con el Puerto Rico Hospital Council y con la Asociación Méca de Puerto Rico, prestará toda clase de asistencia médico-quirúrgica que requieren los miembros de la asociación que soliciten tales servicios del hospital...; se compromete además a prestar toda clase de asistencia postoperatoria hasta el total restablecimiento de los pacientes tratados."

Muy poco comentario requiere esta cláusula ya que, como dijimos en párrafos anteriores, la Facultad Médica de un hospital tiene gobierno propio y la Junta de Directores del hospital no tiene el derecho de firmar contratos con ninguna organización que de manera alguna obligue a la Facultad Médica a prestar servicios de una manera o de otra a menos que no se tenga el consentimiento escrito y previa consulta con esa Facultad Médica.

Creemos que todo contrato que ha formalizado la Cruz Azul

de Puerto Rico con hospitales, los cuales no hayan consultado a su Facultad Médica, queda por lo tanto necesariamente nulo ya que no está dentro de los atributos de la administración del hospital hacer tal cosa.

Realmente no entendemos como se ha podido permitir que hospitales, dirigidos por médicos creyentes en los más altos principios de la Etica Médica, hayan podido firmar contratos de esta naturaleza con organizaciones de seguro de salud voluntario.

Pasando al contrato de la Cruz Azul con los médicos, encontramos que una parte del contrato dice:

> "The physician binds himself not to join or engage in any new agreement or similar contract for medical services with any other association, corporation or groups for the purpose of providing professional services to a group or an association during the life of this contract."

Solamente merece esta cláusula el comentario que de acuerdo con los principios que estableció la Asociación Médica Americana para conceder su aprobación a planes de servicio médico voluntario, específicamente excluía de la lista de aceptación todos los planes que incorporan una cláusula de esta naturaleza en su contrato.

Resumiendo, podemos decir que es nuestra considerada opinión que la Cruz Azul no está autorizada a vender servicios médicos en la isla de Puerto Rico y que esto debe aclararse definitivamente, pidiendo una opinión en cuanto a este punto específicamente, del Procurador General de Puerto Rico. Asumiendo que la Cruz Azul de Puerto Rico esté autorizada a hacerlo, la manera como ha llevado a cabo esta función no ha sido satisfactoria para la Clase Médica o el paciente ya que involuntariamente, estamos seguros, al tratar de llevar a cabo su cometido ha olvidado algunos de los aspectos básicos y primordiales que gobiernan las relaciones entre médico y paciente; y por los cuales es nuestro deber velar para que se mantengan siempre puros e inviolables.

Debemos recordar que la Cruz Azul de Puerto Rico es una **agencia de los hospitales** y no una agencia de los médicos. El Artículo #24 del reglamento, promulgado por el Superintendente de Seguros de Puerto Rico en enero de 1949, establece que la "**responsabilidad económica** por servicios a los suscritores" será asumida por los hospitales asociados en cualquier momento a través de contratos que se hagan con las asociaciones.

La opinión del Hon. Procurador, antes mencionada en este informe, también deja establecido que la "atención de hospital" será provista por "algún hospital o grupo de hospitales", y sí se de-

finió que esto incluía **servicios médicos** o profesionales, entonces es claro también que la responsabilidad final por los **servicios de hospitalización** y los **servicios médicos** que hoy día presta la Cruz Azul recae en los hospitales asociados; y que por consiguiente, son los hospitales los que están vendiendo, controlando y administrando los servicios profesionales que ofrece este plan.

No necesitamos repetir que este tipo de arreglo está en contra de por lo menos cuatro de los cinco postulados de la Asociación Médica Americana, y los cuales enumeramos anteriormente, en las páginas 5 y 6 de este informe.

VI. Conclusiones

Los planes de servicio médico voluntario han demostrado su verdadero valor y no debe quedar la menor duda en la mente de los aquí presentes que estos planes seguirán desarrollándose y que han de constituir un factor importantísimo en la distribución de los servicios médicos en los Estados Unidos y Puerto Rico durante muchos años venideros.

Es por lo tanto la responsabilidad de la Asociación Médica de Puerto Rico asumir una posición clara, firme y definitiva en relación a la manera como los servicios médicos voluntarios han de distribuirse en nuestra isla, teniendo en mente el solo propósito de velar porque el mayor número de personas sean beneficiadas y que el beneficio médico a recibirse sea el máximo y a tono con los adelantos de la medicina moderna.

Estamos obligados a asumir este liderato ya que es la única manera de asegurar a nuestros pacientes que recibirán servicios médicos organizados, controlados y dispensados exclusivamente por la profesión médica de Puerto Rico.

La Asociación Médica de Puerto Rico no estará en el negocio de seguros sino más bien estará contribuyendo a que mejor calidad y mayor cantidad de beneficios médicos sean repartidos entre nuestros ciudadanos. Es importantísimo recordar que dependiendo del interés que nuestra asociación preste a este asunto, el futuro de la Medicina en Puerto Rico depende ya que si no recogemos el guante y nos enfrentamos al problema, dándole nuestro decidido respaldo y entusiasta cooperación, brindaremos las oportunidades para la intervención del gobierno y el control de la Medicina por agencias gubernamentales.

Los médicos que asumen la actitud de que estos planes serán de corta duración o vida deben desechar de su mente esta idea o prepararse para un rudo despertar. Estos planes tienen raíces permanentes en el campo de la medicina moderna y somos nosotros quienes debemos instrumentarlos, dirigirlos y controlarlos. El de-

sarrollo feliz de estos planes ha de depender en gran parte de la honestidad profesional de los médicos participantes para que no se cometan abusos; y debe ser la Asociación Médica el factor determinante quien impondrá la disciplina necesaria entre nuestros miembros para que respeten los principios de estos planes y se logre un éxito total en su establecimiento y desarrollo.

Por otro lado, los pacientes ayudarán a limitar cualquier abuso según vayan siendo enseñados que el costo del servicio médico voluntario está determinado directamente en una gran parte por la honradez con que use el suscritor sus beneficios.

Los planes de servicio médico voluntario en nuestra isla, para llegar a rendir el máximo de los beneficios en los objetivos médicos para lo cual han de crearse, deben gozar necesariamente del absoluto respaldo de la medicina organizada en Puerto Rico, o sea la Asociación Médica de Puerto Rico, y los médicos entender que nuestro futuro está íntimamente abrazado al futuro de estos planes de servicio médico, y su fracaso una vez establecidos significará el colapso total del movimiento de seguro médico voluntario.

Todos los médicos deben compenetrarse íntimamente con los principios de los planes de Escudo Azul y darle su sincera ayuda si es que con la más pura conciencia quieren desempeñar la responsabilidad que la comunidad ha depositado en ellos. Una vez que queden establecidos en nuestra isla tenemos que aceptar sin reservas mentales que ellos han de ser la expresión de la Asociación Médica de Puerto Rico, y este endoso nuestro es el único que puede llevarlos a su éxito y completa madurez.

Nuestro plan ganará rápidamente su puesto en nuestra comunidad y estamos llamados a infundir la confianza y entendimiento mutuo entre los suscritores para que estos también depositen su confianza en nuestro plan, que es el plan de los médicos, que es el plan que habrá de representar el sentir de la Clase Médica de Puerto Rico. Nuestro pueblo desarrollará confianza en nuestro plan de servicios médicos porque el pueblo tiene plena confianza en sus médicos y no puede permitir que esta confianza nunca los abandone.

Por su importancia vital en el desarrollo futuro de la medicina en nuestra isla, el plan de Escudo Azul merece el respeto, el endoso y el liderato de la Asociación Médica de Puerto Rico, y debemos actuar con el único y primordial interés de que nuestros pacientes estén cubiertos, sin importarnos que otras organizaciones vendan seguros de salud, siempre y cuando lo hagan de acuerdo a los principios que ya hemos mencionado.

Es nuestra opinión que la competencia, entre organizaciones, por el seguro de salud es beneficioso para el pueblo, y solo nos brindará una magnífica oportunidad para demostrar, sin lugar a

dudas, que el plan operado por los médicos es el mejor y el que más adecuadamente puede servir las necesidades médicas del suscritor. Eventualmente, los competidores tendrán que retirarse del campo y no ofrecer planes de seguros que no tendrán nunca el endoso de la medicina organizada.

Ya que no se pueden establecer en Puerto Rico organizaciones con fines no pecuniarios para rendir servicios médicos sin incluir servicios de hospitalización al amparo de la Ley #152, según la opinión emitida por el Procurador General de Puerto Rico, el método para establecer el plan de Escudo Azul deberá ser el siguiente:

1. Organizar una compañía mutua y al amparo de la Ley General de Seguros de Puerto Rico. No hay necesidad de legislación especial para la creación de un plan de seguro médico como el que recomendamos.
2. Todo plan de servicio médico que se organizara al amparo de la Ley #152 tendría que ser una duplicación de la Cruz Azul de Puerto Rico y no estaría bajo ningún concepto auspiciado o controlado por la Clase Médica de Puerto Rico.
3. No es posible el establecimiento de un plan de seguro médico controlado por la Asociación Médica de Puerto Rico y administrado en su totalidad o en parte por el personal administrativo de la Cruz Azul de Puerto Rico.
4. El plan de Escudo Azul debe tener el endoso incondicional de la medicina organizada de Puerto Rico.
5. Este plan tiene que ser organizado, controlado y operado por la Asociación Médica de Puerto Rico y llenar todos los requisitos de aprobación, postulados por la Asociación Médica Americana.
6. Para evitar la interferencia indeseable de otros intereses, o la influencia y dominación de los hospitales, los médicos deben mantener un absoluto control de todas las fases y actividades del plan.
7. El plan debe estar administrado por personal competente en este ramo, delegando los médicos la administración pero reteniendo el control y la responsabilidad final.
8. El plan básico debe proveer beneficios en forma de servicios médicos totales, con un límite pre-determinado o "ceiling" de ingreso anual por paciente. Sobre esta cantidad anual, el plan no debe proveer beneficios totales en forma de servicio médico. El ingreso anual que ha de servir de límite a los beneficios médicos totales, debe ser un número realista y suficientemente alto para poder cubrir a todas aquellas personas de ingresos marginales, limitados e insuficientes para enfrentarse a gastos extraordinarios.

9. Los beneficios en forma de servicios médicos satisface mejor el deseo de los suscritores de entradas limitadas y ayuda grandemente a elevar la posición del médico ante el público, ya que no enfatiza el factor de dinero sino el factor de servicio médico.
10. Beneficios en forma de servicios médicos provee la única incontrovertible justificación para que los médicos asuman control de un plan de seguro de salud, y proveen al médico la oportunidad de ofrecer algo que sólo él puede ofrecer, ayudando en esta forma a mantener nuestra libertad profesional.
11. Solamente un plan nuestro podrá combinar factores, tales como "deducibles", "co-aseguro", "pagos en efectivo", y "beneficios de servicios médicos", en un plan armonioso y aceptable para la profesión y para el público.
12. No existe razón alguna para que un nuevo plan pueda cometer graves errores, pues todos los errores concebibles han sido ya cometidos por uno u otro de estos planes. El uso prudente de la información y experiencia adquirida por los planes de Escudo Azul en funcionamiento, evitará que nuestro plan cometa errores básicos de criterio y evitará los problemas que otros planes han tenido que resolver durante los últimos 13 años.

VII. Recomendaciones

Tomando en consideración lo arriba expuesto, este comité recomienda a los miembros de la Cámara de Delegados de la Asociación Médica de Puerto Rico lo siguiente:

1. Que se organice inmediatamente, por la Asociación Médica de Puerto Rico, una compañía mutua, para ofrecer servicios médicos voluntarios pre-pago, con el asesoramiento de los consejeros legales de la Asociación y la cooperación de la Oficina del Superintendente de Seguros de Puerto Rico.
2. Que se haga una asignación de fondos para obtener los servicios de un actuario consultor que prepare los detalles de funcionamiento del plan.
3. Una vez preparado los detalles del plan, este debe ser sometido a esta Cámara de Delegados para su aprobación y luego someterlo a la consideración de una asamblea general de la Asociación Médica de Puerto Rico.
4. Que se nombre por la Cámara de Delegados un comité de 3 miembros, quienes serán responsables de la organización inicial y funcionamiento del plan y quienes tendrán de conseje-

ro al actuario consultor, hasta tanto se pueda asegurar razonablemente la estabilidad futura del plan.

5. Que por ser de tan vital importancia y trascendencia este problema, se tome acción inmediata y definitiva. Estamos destinados a tomar una decisión crucial en este momento, y si actuamos prudentemente, con orientación clara e ideales elevados, será nuestra la recompensa de ver que los más nobles y sanos postulados de la profesión médica han sido defendidos y que nuestros pacientes han recibido plenamente los amplios beneficios de la medicina moderna, que nosotros, y solo nosotros, podemos ofrecerles.

E. Colón Yordán, M.D.
Presidente, Comité Especial
de Escudo Azul

FUENTES DE INFORMACION

1. *Law of Hospital, Physician and Patient* - Hayt, Hayt, and Groeschel.

2. *The Health Insurance Story* - Health Insurance Council.

3. *The Growth of Voluntary Health Insurance* - A.M.A., Council on Medical Service.

4. *The extent of Voluntary Health Insurance Coverage in United States.* - The Health Insurance Council.

5. *Voluntary Prepayment Medical Benefit Plans* (1954) - A.M.A., Council on Medical Service.

6. *Medical Economics* (varios artículos recientes publicados, en que varias autoridades discuten asuntos de la Cruz Azul y el Escudo Azul).

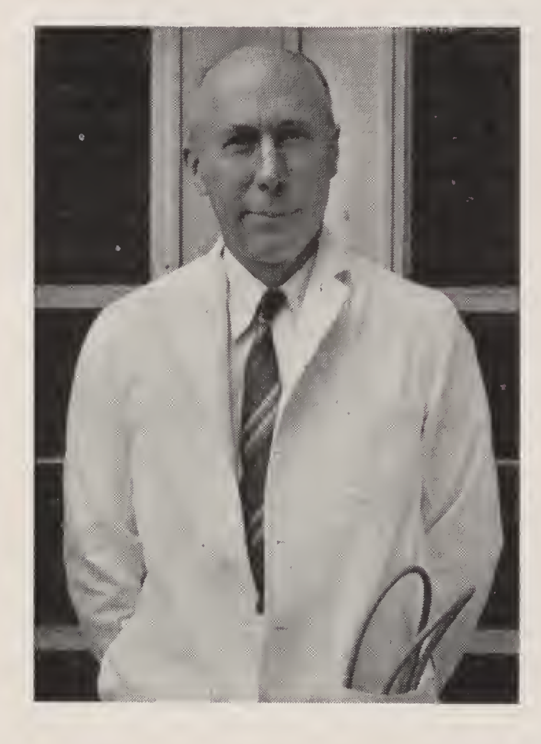
7. *Reglamento promulgado por el Superintendente de Seguros de Puerto Rico*, 28 de enero de 1949 - para la autorización y funcionamiento de asociaciones con fines no pecuniarios para prestar servicios de hospitalización.

8. Entrevista personal con el Sr. M. Nieves Hidalgo, Superintendente de Seguros de Puerto Rico.

9. Información obtenida de los directores de cerca de 40 planes de Escudo Azul en operación en Estados Unidos.

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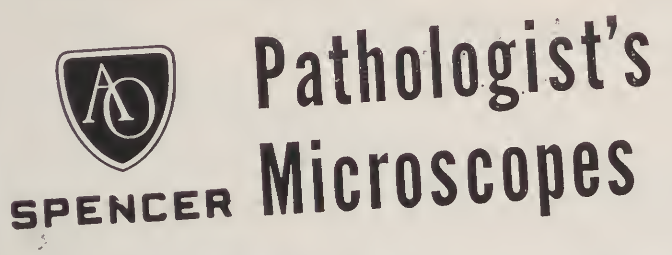
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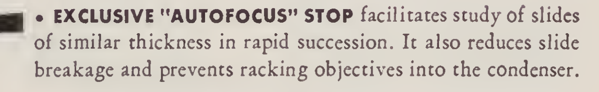
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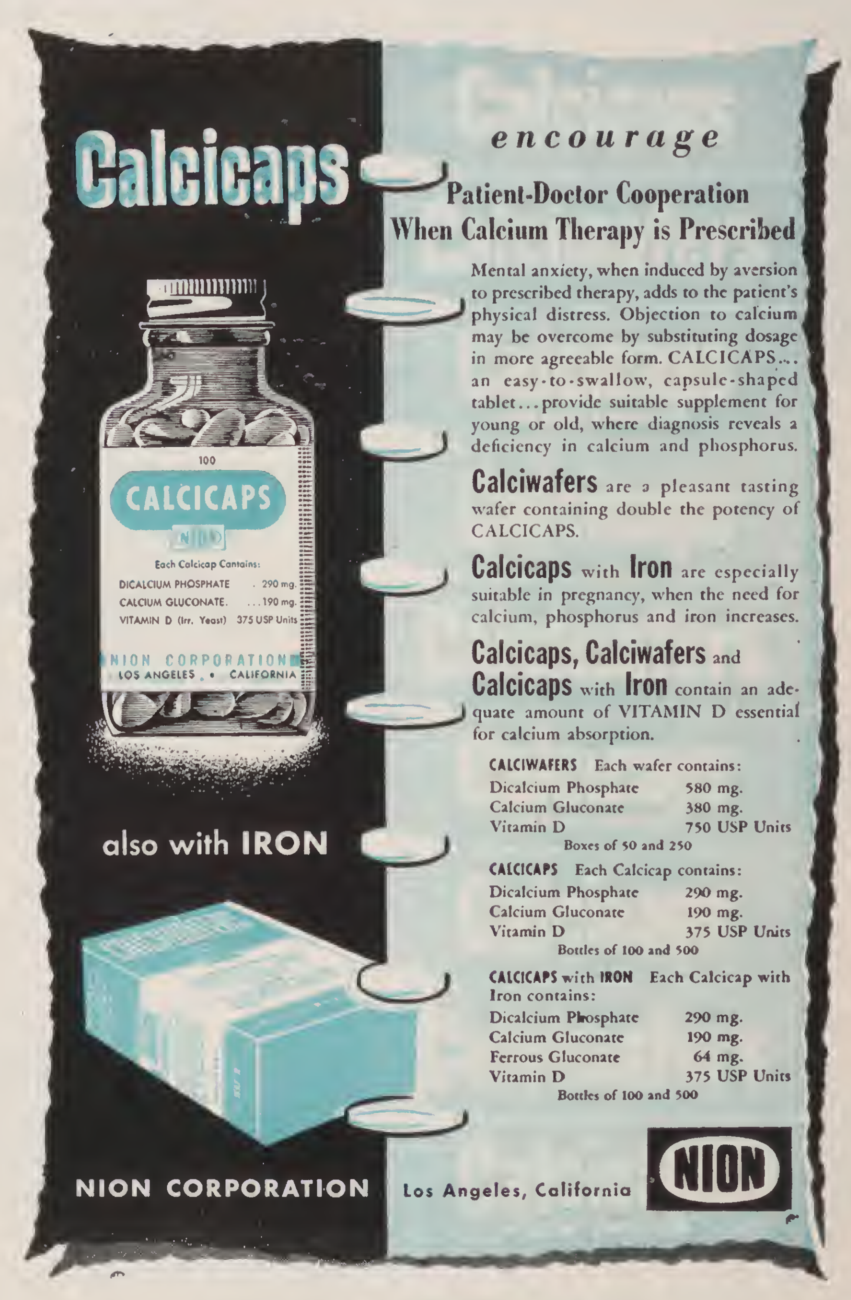
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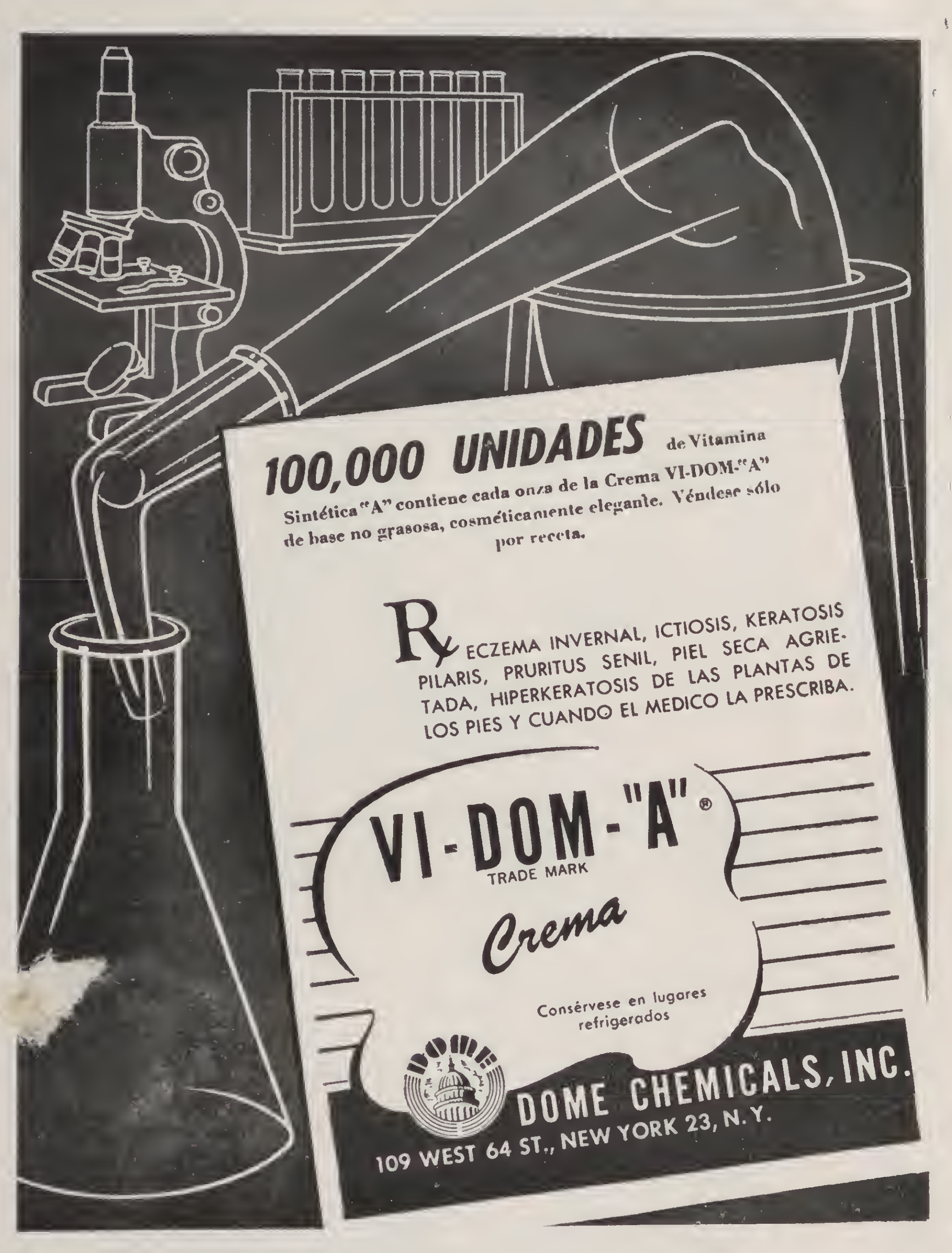
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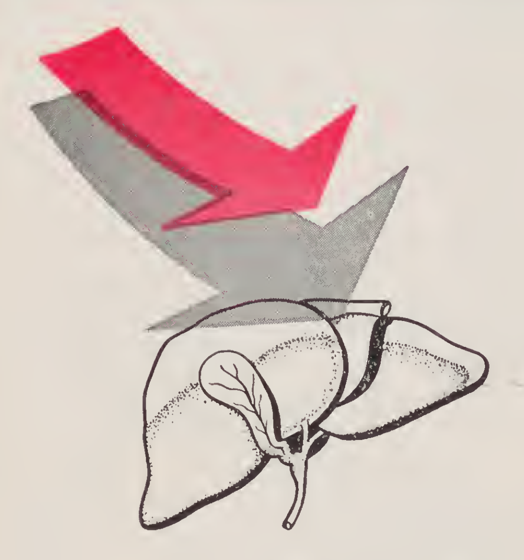
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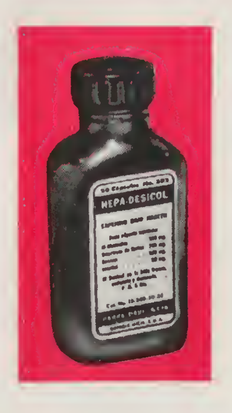
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DE LA ASOCIACION MEDICA DE PUERTO RICO

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SPLENIC VENOGRAPHY

HECTOR F. RODRÍGUEZ, M.D. and RAFAEL DÍAZ-BONNET, M.D.***

Angiography has become an accepted and widely utilized diagnostic procedure. Cerebral angiography, phlebography, angiocardiography and aortography are important adjuncts in the diagnosis of vascular abnormalities. More recently, portal venography has gained recognition as a valuable aid in the study of the portal circulation. Two techniques are used in the roentgenologic visualization of the portal system: 1) splenic portography, in which radiopaque material is injected directly into the spleen, and 2) portal portography, performed at the time of laparotomy, the dye being injected into the portal system through the superior mesenteric vein. Although Rousselot and his group[1] suggest that both procedures be performed in cases of portal hypertension prior to surgery, our results to date have shown that splenic venography alone usually gives the necessary information.

During the last year, 35 attempts at percutaneous splenic portography were carried out at the Bayamón District Hospital, Bayamón, P. R., 28 of them being successful (80%).

Procedure: After ruling out sensitivity to 70% Diodrast by performing an intravenous test, the patient is placed in the supine position on the X-ray table. Two per cent novocain is used as local anesthetic. The site of injection is either the ninth intercostal space in the midaxillary line in non-palpable spleens, or the left upper quadrant at the left anterior axillary line (subcostal approach) in enlarged spleens. With the patient holding his breath, and using a 3-inch, 18-gauge needle on a 50 ml. syringe, 25-30 ml. of 70% Diodrast are injected as rapidly as possible (usually in 5 seconds) directly into the spleen. An X-ray film is taken at the end of the injection.

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Figure 1. A normal splenoportogram is illustrated. Radiopaque material remains in the spleen. The splenic and portal veins have a smooth course and a normal caliber. The intrahepatic branches are clearly visualized.

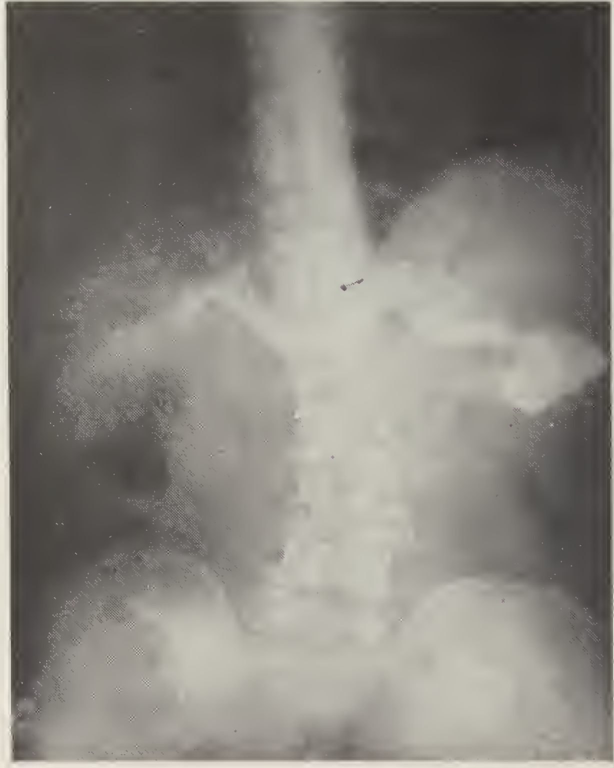


Figure 2. The splenic and portal veins are moderately increased in caliber. The arrow points to the coronary vein, arising from the portal vein. The intrahepatic branches are fairly well outlined. This is a case of portal cirrhosis with portal hypertension.



Figure 3. The splenic and portal veins are greatly distended and tortuous in their course. The intrahepatic branches are not filled well, suggesting intrahepatic obstruction to portal flow. This venography depicts a case of portal cirrhosis with portal hypertension.

Results: A normal splenoportogram is illustrated by figure 1. The splenic and portal veins have a normal caliber and a smooth course. The intrahepatic branches are clearly outlined, denoting no obstruction to the portal blood flow. Collateral branches are not present, indicating a normal hepatopetal flow of blood. Hepatofugal circulation (see figure 2) is pathologic and denotes portal hypertension. Some of the dye is seen deposited in the spleen. Absorption of this radiopaque material is complete in six to twelve hours.

Portal hypertension may be diagnosed by splenoportography. The splenic and portal veins are tortuous in these cases, and have an increased caliber (see figure 3). The intrahepatic branches are usually poorly visualized, indicating intrahepatic portal obstruction. As mentioned above, collateral branches are often depicted. The presence of esophageal varices is frequently illustrated by splenic portography. Atkinson et al[2] reported a greater efficacy in demonstrating varices by venography than when barium swallow or esophagoscopy, alone or in combination, were used. It should be

stated, however, that they used serial films, while our series was limited to a single film per case.

It is said that extrahepatic obstruction to portal flow should be suspected whenever portal hypertension is present with relatitively normal liver function tests. In Puerto Rico, portal hypertension is frequently caused by Schistosoma mansoni infestation of the liver (see table 1).[3] In these patients, one may find splenomegaly, esophageal varices and relatively normal liver function tests. By means of splenoportography, extrahepatic obstruction due to portal or splenic vein thrombosis may be excluded. This method is the only one by which extrahepatic obstruction may be diagnosed prior to surgery and the site of the obstruction accurately indicated. Without this roentgenologic support, the surgeon who elects to do a portocaval anastomosis may find, after a painstaking dissection, a thrombosed portal vein. A case of portal vein thrombosis diagnosed preoperatively by percutaneous splenic venography has been previously reported by us in the literature.[4]

TABLE I

INCIDENCE OF ESOPHAGEAL VARICES IN PORTAL AND BILHARZIAL CIRRHOSIS

Portal cirrhosis (69 cases)		Bilharzial cirrhosis (22 cases)		Bilharziasis and alcoholism (11 cases)	
Cases found	Per cent	Cases found	Percentage	Cases found	Percentage
19	27.5	12	54.4	8	72.7

Table illustrating the relative incidence of esophageal varices in portal cirrhosis, bilharzial cirrhosis and cases having both Schistosomiasis and history of alcoholism. (From Rodríguez et al: Gastroenterology: 29: 235: August, 1955)

Splenoportography has proved to be a valuable means in the rational selection of cases for portocaval or splenorenal shunt. Rousselot and his co-workers[5] outline the indicated venous anastomosis as follows: "1—Portal hypertension with moderate splenomegaly, small caliber splenic vein, large patent portal vein, portocaval shunt. 2.—Portal hypertension with marked splenomegaly, large caliber splenic vein plus hypersplenism = splenectomy and splenorenal shunt. 3—Portal hypertension, obliterated portal vein = splenectomy and splenorenal shunt." By performing a splenic venography the indicated shunt can be chosen.

After the venous anastomosis has been performed, it is often desirable to determine the patency of a portocaval shunt. We have accurately established this by postoperative splenoportography

months after surgery. Figley et al[6] have also reported successful results with this method.

Cooper et al[7] and Figley and his group[6] have recommended the use of splenoportography in patients with carcinoma of the pancreas prior to surgery, to demonstrate involvement of the splenic and portal veins. We have no experience as yet in this phase of splenic portography.

Undesirable effects have been negligible up to the present time. Warmth of the body, flushing of the face, nausea and vomiting, usually are the only complications encountered. A few of the patients complained of pain at the site of injection, which lasted for about 10-15 minutes. Only one of the 35 cases bled appreciably from the splenic puncture. At the time of operation 150 ml. of blood were found in the peritoneal cavity, although no active bleeding was present when laparotomy was performed. In spite of the relative safety of this procedure, we feel it should be undertaken in institutions with skilled surgical teams who can perform an emergency splenectomy if active bleeding occurs.

SUMMARY

1—Percutaneous splenoportography is a valuable adjunct in the study of portal hypertension. Its uses are varied: it indicates the presence of increased pressure in the portal system by showing tortuous and greatly widened splenic and portal veins. Frequently, collateral veins are depicted. Esophageal varices are often demonstrated. By showing the relative caliber of the veins, the ideal shunt operation is selected. Splenoportography indicates whether the obstruction producing portal hypertension is within the liver or without. The patency of a portocaval shunt may be established after surgery. Some authors have recommended its use in patients with carcinoma of the pancreas prior to surgery, to demonstrate involvement of the splenic and portal veins.

2—The technique has been found to be simple and reasonably safe. However, it has been emphasized that the procedure should be undertaken in hospitals with adequate surgical facilities so that an emergency splenectomy may be performed if active bleeding from the splenic puncture occurs.

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HEPATIC TESTS*

*RAMON A. SIFRE, M.D.***

The liver is an organ of many different functions. In hepatic disease some of them, all of them or none of them may be disturbed. The disturbance in these functions is determined by the type of disease affecting the liver and also the stage of this disease. Furthermore different patients show different degrees of disturbance and different functions affected at the same stage of the same disease. For these reasons we cannot expect any one of the hepatic tests to be adequate in providing information as to the status of the liver in all instances. Therefore it becomes imperative to use a group of tests in any case suspected of harboring liver pathology. This is particularly important in the detection of early or mild hepatic disturbance which may at times be only reflected by one of a number of tests. Furthermore frequent repetition of a selected group of tests is important, since their results may vary from week to week and the nature of their change may provide us with helpful information as to the diagnosis, course and prognosis of the patient in question.

We must also realize that the tests at our command are most often not specific for hepatic disease and that other conditions may affect them, e. g. the cholesterol in thyroid disease or the alk. phosphatase in bone disease.

The diagnostic value of hepatic tests thus depends on their proper interpretation in the light of the history, physical examination, the stage and the course of the disease, the results of other procedures and the presence of other ailments which may alter their results. However when properly chosen, performed and interpreted they may be of great value to us.

We use hepatitic tests as helpful aides in:

(1) The differential diagnosis of jaundice.

(2) In the absence of jaundice to establish the presence of hepatic disease.

(3) In order to determine the severity of hepatic injury and follow the course of illness throughout its duration.

(4) In the evaluation of recovery from liver injury.

It goes without saying that in order to obtain the greatest benefit from these tests a good responsible laboratory is indispensable.

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* Presented before the Puerto Rico Medical Association, Eastern District Convention, August 1955.

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We shall now attempt to discuss the various hepatic tests individually before discussing their importance in relationship to diseases as a whole.

1. **The serum bilirubin**

An evaluation of prompt direct reacting bilirubin may occasionally provide evidence of onset, presence or persistance of hepatic disease when the total sr. bilirubin is within normal limits, thus indicating a disturbance which would not have been manifested by determination of the total sr. bilirubin alone.

In hemolytic disease the indirect sr. bilirubin will rise but the prompt direct reacting bilirubin will remain normal or only slightly increased.

In both parenchymatous liver injury and obstruction of the biliary passages there is usually elevation of the prompt direct reacting bilirubin and thus it is of no value in the differentiation between hepatocellular and obstructive jaundice. Fluctuations in the degree of elevation of sr. bilirubin may be indicative in obstructive jaundice of obstruction due to stones.

2. **Urine bilirubin**

Failure to find bilirubin in the urine in the presence of jaundice suggests the presence of a hemolytic process.

The determination of urine bilirubin is also important in the preicteric period of acute-viral hepatitis when it may be found before any elevation of the sr. bilirubin has occurred.

3. **Urine urobilinogen**

Elevation of urine urobilinogen may be the only indication of hepatic disease. However it may also be elevated in other conditions such as in a hemolytic process or in pulmonary or myocardial infarction. When absent over a prolonged period of observation in the presence of jaundice it is strongly suggestive of the obstructive type of jaundice.

4. **Fecal urobilinogen**

This is a determination which can provide very useful information and which many of us are guilty of not using often enough.

When there is complete obstruction of the biliary passages the amount excreted in the feces is usually less than 5 Ehrlich units per 24 hrs.

On the other hand in hemolytic processes the amount usually exceeds 300 units per 24 hrs. It may also be elevated during the period of clearing following extrahepatic or intrahepatic obstruction.

5. **Cephalin-cholesterol flocculation tests (Hanger Test)**

The reaction apparently depends on qualitative and quantitative changes in the albumin and/or globulin fractions of the serum proteins and is usually associated with parenchymal cell injury.

In viral hepatitis it may become positive before the appearance of jaundice and often remains elevated throughout its course. At times it may become negative before the disappearance of jaundice in the recovery stage. It is often positive in cases of anicteric hepatitis.

The reaction is usually negative in jaundice due to extrahepatic obstruction but may become positive when this is complicated by severe liver injury or by cholangitis. However this usually does not occur early during the course of the disease and therefore it is very often of value in the differentiation of surgical and medical jaundice. It may be negative in viral hepatitis particularly of the so called cholangiolitic type. Neefe et al[1] found on incidents of 20% negative Hanger tests in a large series of viral hepatitis at the Army Hepatitis Center in Bayreuth, Germany. The test is frequently negative in metastatic or primary liver neoplasm. At times other flocculation tests may be positive with a negative Hanger. For this reason the use of other flocculation tests is desirable, whenever possible.

6. **Thymol tests**

Abnormalities of these tests appear to be associated with changes in the protein-liquid relationships occurring with certain types of hepatic disturbance.

Positive thymol tests are often found in viral hepatitis and other types of primary hepatic disease. However they may also be negative in the presence of active liver injury and thus negative tests have no value in exclusion. On the other hand strongly positive thymol tests are almost always an indication of important hepatic involvement.

Thymol turbidity may often remain elevated late in the course of viral hepatitis when other tests have returned to normal, and thus it is of importance in determining residual liver damage.

7. Zinc sulfate turbidity

Mixtures of dilute solutions of heavy metals and human sera yield turbidity, the intensity of which is roughly proportional to the quantity of gamma globulin present. The zinc turbidity test is based on this reaction and thus may be influenced by any process affecting the gamma globulin. We may then, find it elevated in conditions such as rheumatoid arthritic or rheumatic fever without primary liver disease. It is frequently elevated in viral hepatitis and other inflammatory conditions of the liver and may be elevated in the presence of negative thymol and cephalin-cholesterol tests. It is usually not elevated in uncomplicated extrahepatic obstruction and thus it may be of differential diagnostic value. The uppermost limit of normal is 12.5 Units. Popper[2] has emphasized that values of less than 6 units are also of diagnostic value and indicative of cholestasis. These low values are felt to be due to regurgitation of an unknown depression factor of the bile in obstructive jaundice.

8. Serum Protein Partition

Many conditions such as nephrosis, multiple myeloma, etc. may affect the serum proteins. Therefore the significance of these in liver disease depends upon the exclusion of other conditions.

Marked decrease in sr. albumin is often observed in association with prolonged or severe liver damage, occurring most characteristically in active portal cirrhosis.

Normal sr. albumin levels do not exclude the presence of hepatic disease, since they may be present in viral hepatitis or compensated cirrhosis. However when the levels are abnormal, they give us a very good indication of the degree of hepatic involvement.

9. Prothrombin Time and Prothrombin Response

We shall limit our remarks on this test by saying that it is subject to too many variations and therefore it is practically useless in the differentiation of hepatic and posthepatic jaundice as formerly advocated.

10. Total and Esterified Cholesterol

Any process characterized by intrahepatic or extrahepatic obstruction tends to cause an increase in the total sr. cholesterol, at times reaching very high levels. However if this is associated

with severe hepatic parenchymal injury, then the cholesterol may remain normal or only very slightly elevated.

As the liver esterifies cholesterol, in parenchymal hepatic injury there is often a diminution of the concentration of cholesterol esters.

In very severe liver disease such as in acute yellow atrophy there may occur great lowering of the total cholesterol and the ester percentage which may be of prognostic significance.

11. Alkaline Phosphatase

In diseases of the liver and biliary tract associated with obstruction or injury of the extrahepatic or intrahepatic bile ducts the sr. alkaline phosphatase is often increased. It has not been established with certainty whether this is due to interference with the excretion of the enzyme or due to its increased production.

Obstruction of the biliary tract causes the greatest increases in alk. phosphatase. In hepatic diseases the increases are not as marked except for those types in which intrahepatic obstruction with minimal parenchymal injury dominates the picture. Because of these last cases it has only limited value in the differentiation between medical and surgical jaundice.

The following value may be attached to it (1) a normal or only slightly elevated serum bilirubin with a high alkaline phosphatase is common in post-hepatic jaundice and neoplastic disease of the liver but uncommon in diffuse hepatitic disease. (2) the combination of a high sr. bilirubin and normal alkaline phosphatase is common in diffuse hepatic disease and rare in post-hepatic jaundice.

12. Bromulfalein excretion

This is one of the most sensitive indicators of hepatic disease and is particularly useful for the detection of hepatic disturbance in the absence of jaundice. It is very often positive in the preicteric stage of viral hepatitis and frequently reveals persistent dysfunction in the late stages of this disease after the disappearance of jaundice. The test is usually positive in the active phase of hepatic cirrhosis and may be positive when there is primary or metastatic malignancy of the liver. However, a negative test, again does not exclude liver disease.

Since the dye is excreted through the biliary passages after its passage through the parenchymal cells it is of no value in the differential diagnosis of jaundice.

Many other liver functions tests have been proposed at one

time or another in the study of jaundice and hepatic disease. We have limited ourselves to discussing those which are in common usage and which will provide the most useful information.

There is another method by which we can study the liver and which may be helpful in the differential diagnosis of jaundice, that of liver biopsy.

Since many of the hepatic pathologic processes are diffuse in nature, a minute tissue sample will generally suffice to give a representative picture of the entire organ. Aspiration biopsy has clearly defined the histopathology of hepatitis and obstructive jaundice. The differences in the histologic appearance of these two conditions are most pronounced in the first few weeks of the disease. Degenerative changes in the parenchymal cells and an increase in connective tissue develop in long standing biliary stasis and this may render the differentiation between parenchymatous disorders and biliary obstruction difficult. Biopsy is also a valuable aid in the diagnosis of primary or metastatic cancer of the liver. However a negative biopsy does not exclude the diagnosis of cancer. It has also shown great value in Boeck's Sarcoid, schistosomiasis, other granulomatoses and biliary tuberculosis.

Pathologists have repeatedly emphasized that liver tissue near the capsule may normally be distorted in architecture and histologic appearance and also that marked changes in histologic appearance result from prolonged anesthesia and the use of retractors. Therefore, when liver biopsy is to be done at the time of exploratory laparotomy, it is recommendable that the surgeon obtain his biopsy at the beginning of the procedure and also that needle biopsies of the deeper portions of the liver be obtained besides the usual wedge biopsy.

Contraindications

Some contraindications of liver biopsy are as follows:

1. Lack of proper facilities.
2. When the information will not alter treatment or otherwise be of benefit to patient.
3. When exploratory laparotomy is to be done anyway.
4. When possible anatomic abnormalities exist, i.e. colonic interposition.
5. When more than minimal ascites is present.
6. Infection of right lower lobe of lung or pleural cavity.
7. When suppurative disease of biliary tract is possible.
8. When complete obstruction of biliary tract is possible.
9. Bleeding tendency.
10. Uncooperative patient.

Indications

1. When liver disease is suspected clinically but usual diagnostic measures fail to confirm or exclude it.
2. When the nature of hepatic disease is obscure.
3. When the cause of jaundice is obscure and operation may be potentially dangerous.
4. Possible systemic disease with secondary liver involvement not diagnosed by usual procedures.
5. To study effect of therapeutic measures.
6. For advancement of medical science.

The risk of the procedure is minimal, in a recent review by Terry,[3] of 10,000 biopsies reported in the literature, there were 13 deaths, and in all the prognosis had been hopeless. The fatality rate is about 0.12%. However, it is essential that anyone making use of this diagnostic method be thoroughly aware of the risk and the means by which to avoid them, that patients be selected with care and that they be watched and studied carefully before and after the procedure.

Bjornoboe and Iverson[4] investigated the value of aspiration biopsy in cases requiring decision for surgical intervention. Of 1,164 pts. with hepatic disease the decision to operate on 46 was based on biopsy results, all other diagnostic methods having failed to give a clear cut answer. In 4 of them the decision was proved to be wrong at operation, 1 case of cholelithiasis was misdiagnosed as hepatitis and 3 cases of hepatitis had been diagnosed as obstructive jaundice. However, liver biopsy had reduced the diagnostic uncertainty from 4% to 0.5%. Thus we find that the proper use of liver biopsy, combined with a detailed biopsy, careful physical examination and a judicious combination of liver function tests can quite appreciably reduce the element of uncertainty.

Jaundice has been classified by Ducci[5] as follows:

1. Prehepatic
   a. Hemolytic
   b. Nonhemolytic
2. a. Hepatocellular
   b. Hepatocanalicular
3. Post-hepatic
   a. Complete obstruction
   b. Incomplete obstruction

In prehepatic jaundice the diagnosis is seldom difficult. In the hemolytic type the usual findings are characteristic.

1. Presence of anemia.
2. Increase in total sr. bilirubin with normal or only slight elevated prompt direct reacting bilirubin.

3. Absence of bilirubin in the urine.
4. Increase in fecal urobilinogen.
5. Increase in urine urobilinogen.

In the absence of complications prehepatic jaundice is associated with normal hepatic tests. In the chronic forms hepatic injury may occur and then careful study may be necessary in order to demonstrate a hemolytic factor. Complicating biliary obstruction may appear secondary to the high incidence of gallstones in hemolytic disease.

In hepatic jaundice we may have the manifestations of the phenomena of parenchymal injury or of biliary obstruction or of both.

However in the typical case of hepatic jaundice we will find as follows:

1. elevation of both the total and prompt direct reacting bilirubin.
2. strongly positive flocculation tests.
3. normal cholesterol with decreased cholesterol esters.
4. alkaline phosphatase less than 10 Bodansky units.
5. low sr. albumin and perhaps high sr. globulin.

In post-hepatic jaundice the typical laboratory picture is as follows:

1. elevated total and direct sr. bilirubin.
2. negative flocculation tests.
3. elevated alk. phosphatase.
4. elevated total cholesterol with a normal cholesterol ester %.

The larger group of instances of jaundice will follow these typical patterns and in them the diagnosis will be rather easy.

There is smaller group where the diagnosis is very difficult and a few in which it may be impossible to reach.

In extrahepatic obstruction jaundice as the disease progresses or when complications arise such as cholangitis or cholangiohepatitis there will be positivity of the hepatic tests on the obstructive side and also on the hepato cellular side. Therefore early study of the patient is important. In cholangitis the presence of chills, fever and leukocytosis may help us in achieving a correct diagnosis.

In the hepatocanalicular type of jaundice we may find laboratory evidence pointing towards an obstructive jaundice indistinguishable from that produced by extrahepatic obstruction. We must remember that there is no hurry in effecting surgical intervention. There is only one type of jaundice requiring rapid surgical intervention and that is when there is obstructive jaundice associated with cholangitis and rapidly developing evidence of severe liver damage. In most other cases we can afford to undergo a reasonable

period of watchful waiting. The majority of cases of hepatocanalicular jaundice will show diminution of the obstructive dysfunction in a period of 4.6 weeks together with progressive evidence of hepatocellular dysfunction. Thus a period of observation by itself is a very valuable diagnostic aid.

There are **certain corollary** tests that may help us in the differentiation between surgical and medical jaundice as follows:

1. Oral cholecystography
2. sr. amylase and lipase
3. Roentgenographic studies
   a. radiopaque gallstones
   b. changes in stomach and duodenum
4. Papanicolau studies
5. Biliary drainage
6. Positive occult blood test in stools.
7. Needle liver biopsy
8. Non-operative cholangiography
9. Splenoportography

In conclusion we might say that:

1. There is no single hepatic test that is always diagnostic, therefore a combination of tests is necessary.

2. Hepatic tests must be interpreted in the light of the history, physical examination, the stage and the course of the disease, the results of other diagnostic procedures and the presence of other ailments which may alter their results.

3. Negative hepatic tests do not exclude liver disease.

4. Liver biopsy is a valuable adjunct in the diagnosis of liver disease, of systemic diseases affecting the liver and in the differential diagnosis of jaundice.

5. Most cases of jaundice fall into a typical pattern and can be easily diagnosed.

6. Hepatic tests are most helpful during the early course of jaundice before complications have clouded the picture.

7. A period of watchful waiting is often a valuable diagnostic aid.

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DISTANT METASTASIS FROM CARCINOMA OF THE CERVIX

REPORT OF A CASE

*EDWARD O'NEILL, M.D.**

Carcinoma of the cervix spreads by continuity, by the lymphatics and by the blood stream. Direct extension and lymphatic spread have been stressed in the literature. Recently the vascular spread is being re-emphasized.[1-2]

De Alvarez reported a study of 71 cases with primary carcinoma of the cervix all of whom showed evidence of remote metastases. He found the spine to be the most common site for bony metastases, with an incidence of 26.8%, to the femur and to the fibula, with 2.8% incidence each and to the tibia with 1.4%. He recommends a more thorough study of these cases, and radiologic investigation of the chest, spine, long bones, and colon.

Cosby[4] found distant metastases in 17 out of 27 cases of advanced carcinoma of the cervix autopsied.

Ford[6] stated that the presence of metastases in bone from carcinoma of the uterus or cervix often is overlooked because of complicating circumstances which may account for pain. Metastases to the extremities are often mistakenly attributed to inflammatory destructive lesions. Of 14 cases with skeletal metastases, 7 showed relief from pain by irradiation.

Henriksen[10] stated that the frequency of distant metastases in cases of carcinoma of the cervix is not widely appreciated. He found distant metastases in 32.5% of 154 non-treated cases and in 37.8% of 202 treated cases of carcinoma of the cervix. The liver was the most frequent site of metastases, with the bone, lung and bowel involved in that order. The vertebrae and ribs were the most common sites of bony metastases. He reported 0.8% incidence of metastases to the tibia in the non-treated cases and a 1.2% incidence in the treated cases.

CASE REPORT

M.B., 58-year old, white, single female, was first seen on Jan. 5, 1955 at the tumor clinic of St. Joseph's Infirmary, Atlanta, Ga., with the chief complaint of recurrent and not progressive vaginal bleeding of two years' duration every two or three weeks for five to seven days. No serious illnesses, no operations.

Review of Systems: Negative except for nocturia 5 to 6 times;

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catamenia: 14 yrs. every 28 days for 7 days, menopause age 50. Extremities: Painful right knee requiring use of crutches for 3 months.

Physical Examination: Blood pressure 198/120, pulse 108, respiration 30, temperature 99, weight 140 lbs. She was a well-developed, well-nourished white female in no acute distress. Neck: Trachea on the mid-line, thyroid not palpable. Head: negative. Breast: Atrophic, no masses or tenderness. Lungs: Clear to A & P. Heart: Regular rhythm; systolic murmur at the aortic area, soft diastolic at apex. Abdominal: Kidneys palpable but not enlarged. Liver at the right costal margin, smooth. Spleen not palpable. Abdomen relaxed, no abnormal masses. Extremities: 4 plus pitting edema of the right foot, leg and knee, which was tender and fluctuant. Neurological: Negative. Pelvic: Speculum examination revealed an ulcerated lesion, mainly of the posterior lip of the cervix which bled easily on touch. Bimanual examination revealed atrophic external genitalia, and induration of the vagina almost up to the introitus. The cervix was hard and fixed with induration extending into the parametrium, up to the pelvic wall, bilaterally. The uterus seemed to be hypertrophic about twice the normal size and firm.

Clinical Impression: Carcinoma of the Cervix, stage 3. Biopsy of the lesion was reported as "epidermoid Carcinoma, Gr. II". AP and Lateral views of the right knee showed the presence of an extensive bony destructive lesion, involving the upper 7 cm. of the tibial shaft, the upper cm. of the fibula, the distal 2 cm. of the femoral condyle, doubtless representing extensive bony metastatic disease from carcinoma of the cervix. Aspiration biopsy of knee performed on Feb. 22, 1955 was reported as "Metastatic Squamous Cell Carcinoma." X-Ray therapy, from Jan. 31 to Feb. 26, 1955, consisted of 6000 R. to the pelvis and 1317 R. to the knee.

At follow-up visit on March 1, 1955, a Papanicolau smear showed occasional malignant cells. No inguinal or supraclavicular lymphadenopathy was noted. Examination revealed a crater at the apex of the vagina, at the apparent site of the cervix, stenosis of the introitus, and induration of the anterior vaginal wall beneath the bladder, and slight induration on the right but marked and extended induration to the pelvic wall on the left.

Her knee felt better. The Tumor Board recommended no further treatment.

On March 29, 1955, symptoms referable to the knee had improved but she still used crutches. Vaginal examination showed considerable induration of the vault and extension on both sides to the bony pelvis.

DISCUSSION

A case of carcinoma of the cervix with distant bony metastasis to the right knee is presented. This was believed to be an inflammatory lesion until later correctly diagnosed.

At the St. Joseph's Infirmary Tumor Clinic, carcinoma of the cervix is treated with X-ray therapy first and then radium. In this case palliative X-ray therapy only was given.

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LA PREVENCION Y EL TRATAMIENTO DE LAS DIARREAS ANTE LA XIV CONFERENCIA SANITARIA PANAMERICANA

PREPARADO POR LA OFICINA SANITARIA PANAMERICANA

Tal vez el hecho nuevo de observación que más impresionara, en 1954, a la profesión médica y a los interesados en la salubridad continental fué la extrema importancia demográfica de las diarreas y enteritis, en los informes de los países a la última Conferencia Sanitaria Panamericana.[1]

Con distintos nombres y bajo variados procedimientos de registro y clasificación, las diarreas aparecen como la primera causa de muerte en numerosos países y territorios y ocupan uno de los cuatro lugares siguientes en varios de los otros. El cuadro inserto muestra la situación individual de 18 países y 16 territorios que proporcionaron datos para el año de estudio elegido. En algunos de ellos, una proporción importante de las muertes no ha sido registrada o clasificada por causa, aminorando así la influencia efectiva de las diarreas. No obstante esos y otros vacíos, las diarreas constituyen claramente el factor individual más importante de atraso demográfico y una fuente principal de despilfarro económico y de vidas en la mayor parte del Continente.

En la medida en que, una tras otra, varias enfermedades trasmisibles van siendo dominadas —cada vez más hasta interrumpir su transmisión—, se acrecienta la importancia de este grupo de infecciones intestinales, tan complejo en su etiología y patogenia como en los mecanismos de control y prevención. La circunstancia de que varios países hayan visto prácticamente desaparecer las diarreas de sus estadísticas y de sus hospitales, en el lapso de una o dos generaciones, constituye acicate adicional al interés por aplicar las medidas de control conocidas y por estudiarlas mejor.[2-3]

Dos factores en especial, aparte de esta información sobre la gravedad del daño que las diarreas producen, justifican plenamente su alta prioridad en las discusiones de la Conferencia Panamericana[4] y en las preocupaciones de los organismos de salud pública y de la profesión médica. El primero es la aprobación de métodos administrativamente simples para la prevención de las muertes por diarrea, mediante el tratamiento precoz y expedito de los casos. El segundo tiene que ver con la posibilidad mejorada de evitar la enfermedad misma, a través de la acción concertada de los profesionales y servicios médico-sanitarios y de la colectividad en un programa que jerarquice las medidas más eficaces y susceptibles de aplicación en cada sitio.

Se examinan sumariamente a continuación ambos factores, como fueron considerados por la XIV Conferencia. Ciertamente, al igual

que ha ocurrido con otras enfermedades y problemas de salud pública, la acción médico-sanitaria específica gana posibilidades efectivas de controlar las diarreas cuando introduce medidas simples de tratamiento y prevención, compatibles con las potencialidades económicas y con los hábitos de la colectividad servida.

Tratamiento Simplificado de las Diarreas Infantiles.

Considerando que casi la totalidad de las muertes por diarrea ocurren en niños menores de áreas urbanas o rurales sin recursos de atención médica especializada, la XIV Conferencia Sanitaria Panamericana recomendó el esquema de tratamiento simple y económico que luego se reproduce[4].

LUGAR DE LAS ENFERMEDADES GASTROINTESTINALES(a) DENTRO DE LAS CINCO PRIMERAS CAUSAS DE DEFUNCION COMUNICADAS POR LOS PAISES Y TERRITORIOS(b) DE LAS AMERICAS, 1952

Paises	*Lugar*	*Territorios*	*Lugar*
Brasil(c)	Primero	Is. Leeward - San Cristóbal y Nieves	Primero
Colombia	Primero	Puerto Rico	Primero
Costa Rica	Primero	Is. Windward - Dominica	Segundo
El Salvador	Primero	Guayana Británica	Tercero
Guatemala	Primero	Trinidad y Tobago	Tercero
México	Primero	Is. Windward - Sta. Lucía	Cuarto
Rep. Dominicana	Primero	Is. Windward - San Vicente	Cuarto
Venezuela	Primero	Barbada	Quinto
Chile	Segundo	Is. Leward - Antigua	Quinto
Panamá	Segundo	Is. Windward - Granada	Quinto
Bolivia	Tercero	Alaska	—
Honduras	Quinto	Bermuda	—
Argentina	—	Hawaii	—
Canadá	—	Is. Leward - Montserrat	—
Estados Unidos	—	Islas Vírgenes, EUA	—
Paraguay(d)	—	Jamaica	—
Perú	—		
Uruguay(d)	—		

(a) En dos países y en un territorio el grupo de enfermedades intestinales.
(b) Exclusive los países y territorios que no proporcionaron información a la XIV Conferencia Sanitaria Panamericana.
(c) Distrito Federal y capitales de Estado, salvo la ciudad de São Paulo.
(d) Año 1951.

Este no es el único método aconsejable. Su principal valor consiste en ofrecer a los médicos generales, enfermeras y personal auxiliar, un método simple de rehidratación precoz de lactantes y niños con diarrea, cuando no se dispone de las facilidades hospitalarias o de laboratorio, necesarias para un tratamiento especializado, incluyendo el uso de antibióticos, sulfas y otros.

Procedimiento a seguir en el trastorno metabólico en casos de diarreas infantiles.(*)

I. Principios:

1. Tratar o prevenir trastornos de la circulación de la sangre y de la función renal.
2. Reponer las pérdidas de agua y de electrolitos.
3. Mantener un estado de equilibrio mientras se repone gradualmente la alimentación.

II. Casos moderados:

1. Suspender comidas y leche.
2. Ofrecer por vía bucal cada tres o cuatro horas, 25 cc. por kilo de peso de la siguiente solución glucosada electrolítica, diluída en la forma siguiente:

Solución concentrada:

Na Cl (cloruro de sodio)	1.5 gramos
K Cl (cloruro de potasio)	2.0 gramos
H_2O (agua)	15.0 cc.
Jarabe hasta completar	75.0 cc.

Disolver primero las sales en agua hervida y luego agregar el jarabe.

Diluír 5 cc. (1 cucharadita) de la solución concentrada en 60 cc. (4 cucharadas) o la cantidad total en un litro de agua hervida.

Siendo la precocidad de la rehidratación un factor decisivo, los médicos deberán estimular y orientar la intervención oportuna de enfermeras, personal auxiliar y de las propias madres, en muchas regiones. Para este fin, y en ausencia de hospital o de farmacéutico, se obtendrá algún alivio con la siguiente solución casera:

Sal	1 cucharadita nivelada
Jarabe de maíz o azúcar	2 cucharaditas niveladas
Agua	1 litro
Hervir la mezcla	

3. Después de 12 a 24 horas, si el niño orina, bebe la solución con ansia y tiene buen aspecto, reemplazar 1/4 de la solu-

(*) Adoptado del Departamento de Pediatría, Escuela de Medicina de la Universidad del Estado de Luisiana, y presentado a la Conferencia por el Dr. Myron E. Wegman, ex-profesor de esa Universidad y actual Jefe de la División de Educación y Adiestramiento de la Oficina Sanitaria Panamericana.

ción en cada comida con una de las mezclas usuales de leche; o, si se alimenta de pecho, volver a darle el pecho durante uno o dos minutos.

4. Gradualmente, en el curso de 3 o 4 días, la solución debe reemplazarse enteramente por la mezcla de leche o la alimentación materna.

III. En casos graves:

1. Suspender toda ingestión por vía bucal.
2. Infusión intravenosa inmediata, en 30 a 60 minutos, de 40 cc. por kilo de peso de una de las dos soluciones siguientes:
   a. Partes iguales de soluciones acuosas de Cloruro de Sodio al 0.9% y de glucosa al 5%, o
   b. 1 parte de solución sexto-molar de lactato de sodio, 2 partes de solución acuosa de Cloruro de Sodio al 0.9% y 3 partes de solución acuosa de glucosa al 5%.
3. En casos extremadamente graves seguir con plasma o sangre, 20 cc. por kilo de peso.
4. Repetir indicación (2), dando 60 cc. por kilo de peso durante las 4 a 8 horas siguientes.
5. Ofrecer por vía bucal cada 3 ó 4 horas la solución glucosada electrolítica diluída, descrita en el párrafo II, 2.

Si el enfermo no tolera los líquidos pero orina, se le puede dar una solución estéril de

Na Cl (Cloruro de Sodio)	1.5 gramos
K Cl (Cloruro de Potasio)	2.0 gramos
Glucosa	100.0 gramos
H_2O (agua) hasta completar	1000.0 cc.

por instilación intravenosa, 150 cc. por kilo de peso cada 24 horas. Esta solución no puede darse por vía subcutánea. Una solución similar, que contenga sólo 33 gramos de glucosa, puede administrarse subcutáneamente.

NOTA: Las soluciones de potasio, que son importantes para que el paciente se recobre, pueden ser tóxicas si los riñones no funcionan bien. No deben administrarse hasta que se establezca la micción.

6. Durante las primeras 24 horas la ingestión total deberá ser de alrededor de 200 cc. por kilo de peso.
7. La alimentación debe reanudarse según se indica en el párrafo II, 3.

Medidas para la Prevención de las Diarreas y su Importancia Relativa

Estas medidas forman parte del bagaje de conocimientos de todo médico. Sin embargo el interés activo de la profesión en aplicarlas es muy limitado, principalmente a causa de su complejidad económica y cultural. De allí que la Oficina Sanitaria Panamericana ha intentado destacar aquellas medidas que merecen más alta prioridad por su influencia directa en la prevención de las diarreas y por su posibilidad de aplicación en países y áreas de máxima incidencia y mínimos recursos sanitarios.

Se resumen a continuación las medidas contenidas en un documento inédito,[6] preparado a base de las conclusiones de la XIV Conferencia con la asesoría de expertos pediatras, epidemiólogos y bacteriólogos.

1. Saneamiento — Las condiciones ideales de saneamiento están bien definidas en la mente y en las aspiraciones de médicos y de sanitarios, como base ineludible de la salud, el bienestar y el confort. Aquí no se habla de ese ideal, sino de aquellas fases elementales del saneamiento que tienen relación más directa con la prevención de las diarreas, miradas como una real emergencia que son

a) Agua. El factor más importante, en la prevención de las diarreas, de acuerdo con la experiencia de Norteamérica y otros países, ha sido el empleo profuso del agua para el aseo personal y el lavado. Su efecto es disminuir el riesgo de ingerir grandes dosis de gérmenes diluyendo la contaminación presente, sobre todo en los utensilios y en las manos. En ausencia de recursos suficientes y de una contaminación grosera del agua, merece, pues, especial énfasis el suministro de una cantidad suficiente de agua en cada casa, como criterio directivo en los programas comunales y en la educación individual.

b) Disposición de Excretas. Es esencial para la prevención de las diarreas. Mientras se introducen sistemas adecuados en todas partes, la protección de las letrinas contra las moscas y la cubertura o entierro de las heces son las medidas más importantes. Los médicos pueden influír poderosamente para cambiar la actitud de las gentes hacia la disposición de las excretas, indispensable a la generalización de métodos sanitarios modernos.

c) Control de Moscas. Es un factor muy importante, pero muy difícil de controlar, sobre todo en las ciudades, como que depende principalmente de un saneamiento completo, que elimine los criaderos. En la prevención de las diarreas, lo importante es evitar el acceso de las moscas a las excretas (véase b) y a las casas

y los alimentos; en áreas rurales, el alejamiento de los animales de las vecindades de la casa puede ser muy útil.

d) Control de los Alimentos. De importancia en las salmonellosis, representa una fase mucho más avanzada del saneamiento. Además del hervido de la leche, práctica habitual en Latinoamérica, las medidas para impedir el acceso de los animales (ratones, aves) a los alimentos son las más posibles y útiles.

2. Educación higiénica del público. Constituye un elemento indispensable en cualquier programa de prevención de las diarreas, que supone siempre un cambio de prácticas de vida. La prédica reiterada será más eficaz si se concentra al empleo profuso del agua para el aseo especialmente de las manos, el alejamiento de las excretas y de las moscas, la protección de los alimentos sobre todo del niño. Pediatras y médicos generales tienen contínua oportunidad de impartir esta enseñanza, sea directamente, sea inspirando a sus colaboradores y auxiliares y aún, si se interesan, estimulando la preparación de medios visuales de educación simples y económicos. Los escolares ofrecen un campo especialmente accesible a esta educación y de gran valor porque alcanza a grandes grupos y se proyecta en las próximas generaciones.

3. Protección directa del niño. En la prevención de las diarreas, la alimentación materna es de extrema importancia en aquellas áreas de malas condiciones higiénicas, que favorecen la contaminación de las mamaderas. La lactancia prolongada es igualmente, de mayor valor donde las fuentes adecuadas de proteína son escasas. La dieta de la madre deberá ser mejorada, incluso mediante distribución de alimentos por los Centros materno-infantiles, para prolongar la lactancia.

La alimentación artificial o mixta, de preferencia con leche en polvo, requiere instrucciones prácticas reiteradas sobre el manejo higiénico de utensilios y alimentos para evitar su contaminación. Al lactante mayor, se le puede dar proteína en forma de leche en polvo agregada directamente a cualquier cocimiento al momento de consumirlo.

La vigilancia organizada del niño por médico y enfermera, idealmente como parte de los servicios sanitarios, tiene un efecto decisivo en la prevención de las diarreas. Es muy importante enseñar a las madres a reconocer los primeros signos de enfermedad y a consultar antes que la rápida deshidratación agrave al niño.

4. Métodos para mejorar la información existente. Las medidas que preceden resumen el conocimiento hoy disponible en la mayoría de los países y que debería aplicarse para reducir la incidencia de las diarreas. Al mismo tiempo, los médicos tienen la posibilidad de contribuír, en grado muy importante, a conocer mejor es-

te complejo grupo de enfermedades, sobre todo en su aspecto epidemiológico. El mejoramiento del registro de causas de muerte, especialmente en niños y la adopción de una clasificación simple de los trastornos digestivos, de acuerdo con la Lista Internacional[7] son un primer paso importante. A medida que se progresa en la prevención de las diarreas, los estudios locales sobre la influencia relativa de los diversos agentes etiológicos y otros mecanismos de transmisión, de la nutrición y factores asociados, adquieren un valor creciente.

——oOo——

La Oficina Sanitaria Panamericana continuará prestando todo el apoyo posible a programas y estudios nacionales o zonales de este tipo, además de participar en numerosas actividades de higiene materno infantil, saneamiento y salubridad general en muchos países.[8] También en este problema, la organización y perfeccionamiento de los servicios nacionales de salud pública, en cuyo logro la profesión médica tiene un papel decisivo, es el instrumento más adecuado de progreso. Como un primer paso, específicamente dirigido a estimular interés en el grave problema de las diarreas y diseminar la información disponible, la Oficina está promoviendo la organización de una serie de Seminarios, nacionales e internacionales, estudios y programas experimentales de control en áreas urbanas y rurales y el mejoramiento de la enseñanza de médicos y paramédicos por medio de becas y ayuda directa a instituciones docentes. Pero en éste, como en muchos otros campos, la posibilidad real de progreso descansa por entero en los servicios de salud pública y en las profesiones médicas de cada país.

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Dr. Francisco M. Susoni

DISCURSO PRONUNCIADO POR EL DR. A. OTERO LOPEZ EN LA ASAMBLEA ANUAL DEL DISTRITO NORTE EN LA CIUDAD DE ARECIBO EL 18 DE SEPTIEMBRE DE 1955, A LA MEMORIA DEL DR. FRANCISCO M. SUSONI

Compañeros y amigos:

Dedicamos este acto a la memoria del Dr. Francisco M. Susoni, por sus relevantes méritos como ciudadano, por sus notables logros en el campo de la ciencia médica y por haber dado a su país todo lo mejor de su vida en su inagotable afán de servir.

El Dr. Francisco M. Susoni nació en el pueblo de Hatillo el 29 de enero del año 1876. Desde niño vino a residir a la ciudad de Arecibo, donde emprendió sus primeros estudios, habiendo cursado su instrucción primaria en la Escuela del Divino Maestro de la noble villa del Capitán Correa. Su instrucción secundaria de Bachiller la realizó brillantemente en el Instituto Provincial de San Juan de Puerto Rico. Estudiante aprovechado y de preclara inteligencia, atrajo para sí la admiración y reconocimiento de sus maestros y mentores.

Inquieto y ambicioso, inquietud y ambición que le distinguieron en el decurso de su vida, acuciado por empeño de superación, ya joven bachiller, se trasladó a la Madre Patria para emprender es-

tudios universitarios. En su mente bullía una idea, solamente una, que al correr del tiempo habría de ser su designio. Su firmeza de carácter, su voluntad y vocación conducíanle a un propósito, el de emular a Hipócrates y llegar a ser digno de su ciencia y abolengo.

En la Universidad de Barcelona cursó los tres primeros años de carrera, pero inquieto siempre, se adentró por tierras de España, y escrutando nuevos horizontes topóse con los de Galicia, y allí en Santiago de Compostela, no sabemos si por acogimiento o porque vislumbrara en lontananza su felicidad futura al encontrar en su ruta por la vida la mujer ideal que más tarde habría de ser su amante compañera, quedóse en las aulas compostelanas y allí recibióse de Doctor en Medicina y Cirugía en el año 1899. Y a esta culminación, débese añadir otra muy significativa, la de haberse unido a la elegida de su afecto.

Casado ya, retornó a la Ciudad Condal, donde permaneció por algún tiempo asociado a uno de sus antiguos profesores y allí hubiese permanecido, a no ser por el precario estado de salud de su progenitor que le llamó a Puerto Rico. Vino con el propósito de llevárselo a Barcelona, para continuar ejerciendo allá su humanitario ministerio, pero la enfermedad de su señor padre se lo impidió y le hizo desistir, permanenciendo en Puerto Rico indefinidamente.

Escogió la ciudad de Arecibo como campo de acción, para desarrollar más tarde múltiples actividades médicas preponderantes y diversas actuaciones públicas ciudadanas que encauzaron su derrotero hacia la cima de bien ganados logros y alcanzados éxitos. Forjado en la lucha, con la energía que brinda la juventud, con aquella inquietud tan propia de su espíritu y con el estímulo de una mente alerta, organizó y estableció, en el año 1903, la primera institución clínico-médica en la ciudad de Arecibo en el sitio que hoy se conoce como Urbanización García. Fué su privilegio. Pero motivos fortuitos, por quebrantos de salud, le obligaron, muy a pesar suyo, de privar a su pueblo de los beneficios que ya empezaba a derivar de este munífico adelanto, viéndose en la necesidad de cerrar su puertas, aunque temporalmente, quizás.

Velando por sí mismo y por el deseo de continuar dando de sí el fruto de sus conocimientos médicos, se fué a París en busca de remedio para sus quebrantos, que consiguió a costa de perseverancia y sacrificio.

En plena rehabilitación de salud, al regresar de Europa, volvió a Arecibo, a la ciudad de sus afectos, a la que tanto quiso, a la que brindó todo su lustre y a la que daría con el pasar de los años toda una vida de éxitos y logros. Fué médico titular y cirujano jefe del Hospital Municipal de Arecibo.

Siempre le interesó la cirugía, a la cual dedicó su mejor contribución y tiempo. Abarcó todos los aspectos de esta especialidad,

incluyendo entre ellos la cirugía ocular, por la cual mostró gran interés. Fué pionero de la cirugía moderna en Puerto Rico. Sus conocimientos anatómicos, su dedicación al estudio de las modernas técnicas quirúrgicas y su natural disposición, hicieron de él un cirujano diestro, sin haber pasado por el tamiz de la escuela postgraduada. Fué clínico de grandes recursos y terapeuta preciso.

En el año 1916, volviendo por sus fueros, fundó la clínica que ostenta su nombre, hoy Hospital Dr. Susoni. En esta institución realizó la más árdua, mejor y más humanitaria labor de su postulado, rescatando con la magia de su bisturí, vidas a la muerte. En ocasiones múltiples fuí testigo de su habilidad en la mesa de operaciones. El maestro nunca se arredraba. Su pericia era inagotable. Su mano era firme. Su conciencia quirúrgica delicada y responsable. Solíame decir, "el vientre es una Caja de Pandora, uno nunca alcanza a comprender las sorpresas que reserva para el cirujano." Y así es la verdad. ¡Cuántos diagnósticos y pronósticos cambian de faz al exponer el campo operatorio!

Por aquel año 1916 empezaba yo mis primeros pasos de médico novel por el sendero escabroso de la inexperiencia. Con buen bagaje académico y limitado sentido práctico, fué el Dr. Susoni mi mejor mentor y maestro en la difícil tarea de acoplarme a estos dos atributos de mi naciente vida profesional. Era mi consultor en los casos de serias manifestaciones clínicas, y con él aprendí lo que no enseñan los libros: el diagnóstico preciso a la cabecera del enfermo. La maestría del clínico competente, sus sabias lecciones y sus atinados consejos fueron para mí fuente de saber, que avivaban mi mente y estimulaban mis conocimientos, para los que habían necesidad y apremio de salud, por sus quebrantos.

Sirvió a su país por más de cinco décadas, no solamente en actividades médicas sino en otras muy distinguidas de índole patriótica, que la historia en su día habrá de proclamar. Nosotros, como médicos, silenciamos este jalón de su vida, aunque reconocemos, como reconoce todo Puerto Rico el valor de sus hazañas.

Ya para el año 1934 su salud comenzó a declinar y ésto le obligó a alejarse poco a poco de compromisos profesionales. En el 1939, colgó su augusta clámide de médico y cirujano de su Clínica, no sin antes dejar tras sí una estela luminosa de médicos jóvenes, que adiestrados por él, hacen hoy honor al protomedicato puertorriqueño, entre ellos Astor, Susoni hijo, Santos, Rodríguez Olmo, Valdivieso y otros. Sin embargo, no se alejó del todo, no podía irse y abandonar su bajel. Era tal su celo por la institución que con tanto afán fundara, que la bondad de corazón que tantos afectos le granjeó, no le permitió renunciar de una vez a lo que era tan

suyo, y permaneció como director y consultor hasta sus últimos días.

Susoni no fué académico, fué más bien didáctico. No obstante, sería prolijo enumerar los trabajos escritos inéditos que presentó en diversas asambleas y congresos médicos, y fué, a pesar de su acervo intelectual, modesto, para superarse aún más en la firme consistencia de su arte y de su ciencia.

Procreó una familia ilustre que hace honor a su nombre y a su estirpe.

Murió en la paz del Señor el día 28 de noviembre de 1954. Hace apenas un año. Se fué al más allá con la satisfacción del deber cumplido. Su nombre y su paso por la vida honran a Puerto Rico. Su historia habrá de imprimirse con caracteres indelebles en los libros de su tierra, para ejemplo y emulación de las futuras generaciones, y nosotros, que hoy reverenciamos su memoria, debemos sentirnos satisfechos de haber cumplido este deber.

Su retrato, que en esta sala se destaca prominentemente, permanecerá por siempre en este recinto como faro de luz que ilumine nuestras vidas.

MEDICAL DIRECTORY OF PUERTO RICO
ALPHABETICAL LIST OF PHYSICIANS

— A —

Abreu-Román, Antonio, Arecibo
Acevedo, Angel Carlos, Ponce
ACEVEDO, CARLOS E., Hato Rey
ACEVEDO, JUAN E., Mayagüez
Acevedo, Norberto, U.A.
Acevedo-Montalvo, J., Patillas
Acevedo-Santos, Enrique, Guayama
ACHECAR, FELIPE J., Santurce
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Acosta, Guillermo, New York
Acosta, Luis R., Río Piedras
Acosta, Raúl R., Río Piedras
Acosta Torres, José J., Caguas
ACOSTA VELARDE, ANTONIO, New (York
ACOSTA VELARDE, JAIME, Vega (Alta
ADAM, ALBERTO, San Juan
AGUAYO, ROBERTO, New York
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AGUILO, OSCAR, Mayagüez
Aguiló Dies, José A., Santurce
ALEMAÑY, CARLOS E., New York
Alfaro, Pedro Juan, Arroyo
Alfonso, Edmundo, Santurce
Allende, Ramón Luis, Ponce
ALONSO, JOSE J., Hato Rey
ALONSO, MIGUEL, Santurce
Alonso-Caiñas, Fidel, Ponce
AlonsoCervantes, E., Corozal
Alonso-Céspedes, T., Isabela
Alonso-Martínez, R., Fajardo
Almonte, Maximiliano, Mayagüez
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ALMODOVAR, RAMON I., Camp Le- (jeune, N.C.
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Alvarez, Flavio, U.A.
Alvarez, Jesús, Fajardo
Alvarez, Rafael, Fajardo
Alvarez, Urselio, Bayamón
Alvarez-Calderón, J., Santurce
ALVAREZ DE CHOUDENS, J., A.F.
Alvarez-Pagán, Milton, Hato Rey
ALVAREZ-POU, LUIS, Santurce

Names in Capitals - Members P.R.M.A.
U.A. - Unknown address
A.F. - Armed forces

AMADEO, JOSE A., Ponce
Amieva, Mederos, Patillas
AMOROS, LUIS A., Río Piedras
ANDINO, AGUSTIN M. de, Santurce
Anduze Menéndez, Julio, Camuy
ANGELL, IVAN M., Mayagüez
Angulo, Edilberto, Trujillo Alto
Anselmi, Jr., Ventura, Peñuelas
ANTOMMATTEI, ARMANDO, Yauco
ANTOMMATTEI, RENE, Guayanilla
APONTE, JOSEPH L., Hato Rey
ARANA SOTO, SALVADOR, Santurce
ARANDES REXACH, E., Bayamón
Arañjo, Juan, U.A.
ARBONA, GUILLERMO, San Juan
ARBONA, JOSE LUIS, Mayagüez
Arbona Figueroa, José A., A.F.
Arcan, Victor M., San Juan
Arena, Nicolás, Río Piedras
Arias, Tomás, Naguabo
Armaiz, Guillermo R. Vega Baja
Armstrong Mayoral Raúl, U.A.
ARMSTRONG-RESSY, CARLOS, (Santurce
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Arrarás, José E., Mayagüez
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Arrigoitía, Enrique M. de, Arecibo
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Avilés-Pérez, Juan, Mayagüez
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AXTMAYER, ROBERTO, A.F.
Axtmayer, Rosa, Río Piedras
Ayala, Angel Luis, Fajardo
AYALA, ANGEL M., Caparra Heights
AYBAR, JOSE A., A.F.
Aybar, Roberto, U.A.
Azuela, Víctor M., Río Piedras

— B —

BABB, DONALD F., Ponce
Badía, José Miguel, Hato Rey
Badillo, José William, Aguadilla
BADILLO-SANTIAGO, RAMON, (Aibonito
Báez, Ignacio José, Vega Alta
Bajandas, Ahmed, Bayamón
BAJANDAS, FRANCISCO J., Hato (Rey
BALASQUIDE, LORENZO A., Ponce
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BARRERAS, JENARO, Caguas
Barreto Domínguez, A., Aguas Buenas
BARTOLOMEI, LUIS, Ponce
Bartolomei-Santaella, René, Ponce
BOSORA DEFILLO, JUAN, Santurce
BATLLE, FERNANDO A., Hato Rey
Bauzá-Hernández, Antonio, San Juan
BAYONET, NATALIO, San Juan
Becerra, Juan Manuel, Ponce
BELAVAL, JOSE S., Santurce
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Bendeck, Taufick, Santurce
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BERNABE, RAFAEL, San Juan
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BERIO-SUAREZ, CARMEN L., San- (turce
BERIO-SUAREZ, FRANCISCO, (Santurce
BERIO-SUAREZ, JOSE, Santurce
BERIO-SUAREZ, MARIA TERESA, (Santurce
Bernal-Cabrero, Delfín, Santurce
BERNAL-ROSA, JOSE F., Santurce
BERNAL Y DEL RIO, VICTOR, (Río Piedras
Bernaola, Ramón, U.A.
BERRIOS, MANUEL B., Caguas
Berrocal, Carlos, U.A.
Berrocal, Enrique, Santurce
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BIAGGI, HIPOLITO, Mayagüez
BIAMON, LUIS L., San Juan
Biascoechea, Diego, Santurce
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Blanco, José, U.A.
BLANCO, RAFAEL A., Lajas
Blanco Lugo, Pedro, Manatí
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Blasini, Francisco, Santurce
Blasini-Rivera, Marino, Santurce
BLASINI-SANTIAGO, RAFAEL, (Santurce
BONELLI, FRANCISCO, Santurce
BONELLI, PABLO M., Río Piedras
BOND, E. M., Caguas
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Boneta, Fernando, Toa Baja
Bonilla Colón, Jorge, San Juan
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BRINZ, ALBERT J., Río Piedras
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BUSQUETS, ANTONIO R., Ponce
BUSQUETS, SALVADOR C., Hato Rey
BUXEDA, FERNANDO L., A.F.
BUXEDA, ROBERTO, Santurce

— C —

CABRERA, JUAN R., Santurce
Cabrera de la Rosa, Fernando, Río (Piedras
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Cadilla-Viñas, Arturo, Bayamón
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CALDERON, AUREO, Santurce
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Cantón, Rafael, Río Piedras

Capella, Amaury, Santurce
Capestany, Roberto, Ponce
Capó, Francisco J., Puerta de Tierra
Carazo, Jorge, Bayamón
Cardona, Arístides, U.A.
CARDONA, GUILLERMO, Ponce
CARDONA, JOSE NESTOR DE, Isa-(bela
CARDONA, JUAN P., Salinas
CARDONA, NESTOR DE, Aguadilla
Carlo, Enrique J., Boquerón
Carlo, Ramón, Río Piedras
CARRASQUILLO, HONORIO F., Pta. (de Tierra
CARRASQUILLO, MODESTO, Hato (Rey
Carrasquillo, Rafael, Guayama
Carrera, Guillermo M., New Orleans
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CARRERA, MANUEL G., Fajardo
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Carro, José A., Bayamón
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CASANOVA-DIAZ, J. R., Hato Rey
CASHION, MARVIN S., Santurce
CASO, JOSE B., Santurce
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CASTAÑER, ALBERTO, Santurce
Castillo López, Miguel A., Barceloneta
Castro, Carlos M. de, U.A.
CASTRO DE CHAFEY, MARISA, (Santurce
Castro de Jesús, R., Caguas
CASTRO SUAREZ, CARMEN, Santur-(ce
Castro de González, Providencia, (Santurce
Causade, José, Santurce
Cerra Quiñones, Domingo, Santurce
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CESTERO, ANGEL R., Santurce
Chafey, David H., Santurce
CHAVES-ESTRADA, JOSE, Santurce
Cheij Kourie, Abraham, Ceiba
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Christian Mejías, Jorge, Aguadilla
Cintrón-Cruz, E. A., Santurce
CINTRON GARCIA, MIGUEL A., (Ponce
Cintrón-Rivera, Angel A., San Juan
Clavell, Iván, Ponce
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CLAVELL, LUIS C., Mercedita (Ponce)
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Coca Mir, Rafael, U.A.
Coello Michel, José, Juana Díaz
Colberg, Giovani, Bayamón
COLBERG-RIOS, HERMAN, Río Pie-(dras
Colberg, Jorge, Puerto Nuevo
Colberg, Pedro Nelson, Camuy
Colberg, Rebecca, Río Piedras
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COLLAZO, PEDRO J., Hato Rey
Collazo, Rafael, Hato Rey
Coll y Cuchí, Víctor, Santurce
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COLON, RAFAEL, Santurce
COLON, RAMON T., Río Piedras
Colón-Betances, Cristino, Caguas
Colón-Bonet, Juan, Bayamón
COLON-DAVILA, J. A., Santurce
COLON-FONTAN, ANGEL B., Naran-(jito
Colón-Lugo, Salomón, Santurce
Colón-Morales, M., Río Piedras
Colón-Ramos, Francisco, Fajardo
COLON-RIVERA, EGIDIO S., Santur-(ce
Colón-Román, J., Mayagüez
COLON-YORDAN, ERNESTO, Ponce
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CORA, MARINA E., Puerto Nuevo
CORDERO, JERAMFEL, Santurce
CORDERO, RICARDO, Santurce
Cordero de Garcés, Ana L., Santurce
Córdova, Fernando A., Santurce
CORNELL, ELMER, Ensenada (Guánica)
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Correa Ayala, Roberto, Arecibo
Cortés, Félix Manuel, Santurce
Cortés, Nelson, Morovis
COSTA MANDRY, OSCAR, Santurce
Costas, Raúl, Santurce
COSTAS DURIEUX, J. L., Ponce
Crespo, A., Bayamón
Crespo, Jorge U., Lares

Crosiar, Donald, Mayagüez
Cruz, Héctor, Bayamón
Cruz Hernández, H., Arecibo
CUCHI COLL, ENRIQUE, Santurce
Cuello, Leovigildo, Santurce
CUEVAS-ZAMORA, R., Hato Rey
Cummings, Luis E., Santurce
Cumpiano, Emilio, Santurce
Cuquerella, Víctor, Santurce
CURBELO, PABLO G., Santurce
Curet, José A., Fajardo

— D —

D'Acosta, Adolfo, Bayamón
Dalmau, Carlos, U.A.
DALMAU, MIGUEL S., Bayamón
Dalton, Ralph E., San Juan
Darío del Nero, Pedro, Río Piedras
Daugherty, John W., Santurce
DAVILA, JOSE E., Río Piedras
DAVILA, BASILIO, Santurce
DAVILA, JULIO E., Santurce
Dávila-Cintrón, Luis, Cidra
DAVILA LOPEZ, JOSE G., Ponce
Dávila Polanco, José B., Santurce
DAVIS, LEONCIO T., Puerto Nuevo
Defendini, Efraín A., Santurce
Delerme, Félix, Río Piedras
Delgado González, A., Aguadilla
DELIZ ROIG, LUIS R., Hato Rey
Deñó, Américo, Jayuya
Devarie Sánchez, Marcos, Vega Baja
DIAZ, ANGELES, Santurce
DIAZ ATILES, ALBERTO, Santurce
DIAZ BONETT, LUIS, Hato Rey
DIAZ BONETT, RAFAEL, Río Piedras
DIAZ CARAZO, JOSE, Santurce
Díaz de Garau, Priscila, Río Piedras
Díaz Márquez, G., Río Grande
Díaz Martínez, Rafael, Manatí
Díaz Marxuach, Euclides, Caguas
Díaz Montañez, Angel, U.A.
Díaz Montañez, Rafael, Cayey
Díaz Pérez, José M., Ponce
DIAZ RIVERA, RURICO S., Santurce
Díaz Romero, J., Manatí
Díaz Romero, R., U.A.
Díaz Santini, Felipa, U.A.
DIEZ GUTIERREZ, EMILIO, Orocovis
DIEZ RIVAS, FEDERICO, Santurce
DOMINGUEZ, ALBERTO M., California
DOMINGUEZ CARLOS, Washington, D.C.
DOMINGUEZ, CESAR, Humacao
DOMINGUEZ, RAFAEL, Utuado
DOMINGUEZ, TOMAS, Guayama
Domínguez- Vinicio, Maunabo
Dones-Román, Rubén, Arecibo
Duarte Mendoza, Pedro, Juncos
DUNSCOMBE, COLBY W., Mayagüez
DUNSCOMBE, WILLIAM C., Mayagüez
DURAND, PEDRO J., U.A.
Duverge, Héctor, Arecibo

— E —

ECHEVARRIA, ARTURO L., Aguadilla
EHRLICH, LASZLO, San Juan
Elías de Alonso, A., Río Piedras
ELLIS CAMBIASO, FEDERICO, Caguas
ESPINOSA, MANUEL, Santurce
ESTELLA, HONORATO, Santurce
ESTRADA, ISABEL, Massachusetts

— F —

FAGOT, GABRIEL, Bayamón
FAS, NAYIP, Cabo Rojo
Febles, Erasto, Río Piedras
Feliciano, Héctor A., Río Piedras
Félix Rodríguez, C.A., Caguas
Fernández, Benigno, Toa Baja
Fernández, Enrique, Fajardo
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FERNANDEZ, MARIO C., Santurce
Fernández, Nelson, Hato Rey
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Fernández Marchante, R., U.A.
Fernández Pedro, C., Hato Rey
FERNANDEZ SARIEGO, G., A.F.
Fernández Valdés, A., Ciales
FERNOS ISERN, A., Washington
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FERRAIUOLI, FRANCISCO A., Santurce
FERRAIUOLI, C. JOSE, Santurce
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FERRER, JOSE C., San Juan
FERRER, RAFAEL E., Cataño
FERRER, REINALDO A., Santurce

FERRER DELGADO, TOMAS, Maya-(güez
FERRER PIÑERO, LILLIANE, Hato (Rey
Figaredo, Alfredo, Guayama
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FIGUEROA, ETERVINA, Santurce
FIGUEROA, LEOPOLDO, Santurce
FIGUEROA, MIGUEL, A.F.
Figueroa Colón, José J., San Juan
Filiberty, Manuel, Aguadilla
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FLAX, HERMAN J., Santurce
Flores Gallardo, A., Río Piedras
FONT, J. H., Puerto de Tierra
FONT CASALDUC, JAIME, Puerta de (Tierra
FONT SUAREZ, VICENTE, Santurce
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Forés, Sally E., Aguadilla
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Fossas, José Rafael, U.A.
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Franceschi, Francisco, San Juan
FRANCESCHI JULIA, J. A., Humacao
FRANCISCO, ROBERTO, Santurce
FRANCO-SOTO, J. A., San Sebastián
FRANK, JULIO E., Hato Rey
FRASQUERI, EDUARDO R., Santurce
Freidinger, Joseph, Utuado
FUENTES, CLAUDE E., New York
FUERTES, JOSE R., Santurce
Fuertes Correa, Margarita, Arecibo
FUSTER, JAIME L., Guayama

— G —

Galíndez Antelo, William, U.A.
GALLARDO, JOSE A., Santurce
GARAU, SAMUEL, Hato Rey
GARCES, JULIO E., Caparra Heights
García, Arnaldo J., U.A.
GARCIA, CELSO RAMON, U.A.
GARCIA, DAVID E., Hato Rey
García, Herminio D., Hatillo
García, Iván H., U.A.
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García, José Ramón, Aguadilla
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García Carrasco, F., Coamo
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García Esteves, Juan, Santurce
GARCIA GARCIA, J., Santurce
García Jiménez, M., Río Piedras
GARCIA LASCOT, EULALIO, San (Juan
García López, Rafael, Santurce
García Madrid, J., Río Piedras
GARCIA DE LA NOCEDA, HILDA, (Río Piedras
García Quevedo, J., Arecibo
GARCIA DE QUEVEDO, LUIS, (Santurce
García Quevedo, Orlando, Arecibo
GARCIA DE LA TORRE, F., (Santurce
García Estrada, Manuel, Santurce
García Galarza, J. A., Santurce
García Malpica, J., Carolina
GARCIA MERCADO, LEOPOLDO, (San Juan)
GARCIA PALMIERI, MARIO R., (Santurce
GARCIA RAMIREZ, OSCAR E., (Aguirre
García Rivera, A. J., Santurce
GARCIA SOLTERO, A., Guayama
GARCIA UBARRI, ANGEL, Guaynabo
García Vélez, Joaquín R., Santurce
García Zamora, A. J., Aguadilla
García Monte de Oca, C., Ponce
GARRIDO CARMONA, M., A. F.
GARRIDO COLLAZO, J., Río Piedras
GARRIDO MORALES, M., Santurce
GARRIGA PRIDA, JOSE E., Río (Piedras
Garriga Prida, Rafael, Santurce
Gatell, Palmira, Santurce
Gavillan Pabón, Pedro, Río Piedras
GELABERT, JOSE A., Ponce
GELPI, WILLIAM P., Santurce
GELPI, WILLIAM R., Ponce
Gelys, Alberto, A. U.
Gianoni, Domingo, Fajardo
Gil, Julio A., Cayey
GIL, RAFAEL A., Santurce
GINORIO, ANTONIO R., Santurce
GLOVER, SAMUEL I., Mayagüez
GODREAU, MIGUEL F., Ponce
GOMEZ ACEVEDO, MANUEL, Río (Piedras
GOMEZ, HIRAM E., E.U.
González, Antonio, Santurce

GONZALEZ, BENIGNO T., Caparra (Heights
González, Bernardino, A.U.
GONZALEZ, CARLOS, Río Piedras
González, Eduardo, Bayamón
González, Francisco, Aguadilla
González, Georgina M., Santurce
GONGALEZ, F. G., Santurce
González, Jacinto, Río Grande
GONZALEZ, J. F., Mayagüez
GONZALEZ, HECTOR M., Humacao
González, Luis F., Río Grande
González, Modesto, Arecibo
GONZALEZ, OSVALDO, Santurce (A.F.)
González Alcover, R., Santurce
González Correa, R., Río Piedras
González Díaz, Manuel, U.A.
González Flores, B., Santurce
GONZALEZ GIUSTI, J. R., Santurce
González Mena, A., Hato Rey
GONZALEZ MONTALVO, Lydia, Río (Piedras
GONZALEZ FLORES, J. R., Ponce
González Olmo, Juan B., Lares
González Orozco, Mario, A.U.
González Ríos, Antonio, Santurce
GONZALEZ RAMIREZ, LUIS, New (Jersey
González Saldaña, L. E., Santurce
GORLIN, PHILIP S., Ponce
GOWE, DONALD F., Ponce
GOYCO, OSVALDO, Ponce
Grana, Julieta, Santurce
GRAULAU, LUIS M., Camuy
GUARCH DE FLAX, JOSEFINA, (Santurce
Guardiola, Pablo R., A.F.
Guerrero Guerrero, R., Río Piedras
GUBERN, CARLOS S., Fajardo
Guevara, Raúl A., Camuy
GUIJARRO, ANTONIO, New Jersey (A.F.)
Gutiérrez, Armando A., Guayama
GUTIERREZ ORTIZ, VICTOR, (Santurce
GUZMAN, LUIS R., Santurce
GUZMAN ACOSTA, CARLOS, (Santurce
GUZMAN ACOSTA, MANUEL, (Santurce
Guzmán López, Manuel, Bayamón
GUZMAN RODRIGUEZ, M., Santurce

— H —

HADDOCK-SUAREZ, JENARO, Río (Piedras
Haddock-Suárez, Lilliam, Santurce
Hammerschmidt, César, Villalba
Hart, Humberto, Río Piedras
HAZIM, HAMLET, Ponce
Heiman, Harold, Santurce
HERETER, JORGE, Hato Rey
HERNANDEZ, RAFAEL, Alabama
HERNANDEZ MATOS, J. A., Río (Piedras
HERNANDEZ MORALES, F., (Santurce
Hernández Sanz, Víctor, Mayagüez
HERNANDEZ DEL VALLE, PEDRO, (Fajardo
HIDALGO, HECTOR O., Santurce
HIDALGO CESTERO, CARLOS A., (Río Piedras
Hinman, Edgar Harold, San Juan
HOMEDES, JUAN, Hato Rey
HONORE, SABAS, Ciales
HOSTOS, EUGENIO M. DE, (Santurce
Hoyo, Manuel, Hato Rey
HOYOS, HECTOR A., Ponce

— I —

Ifarraguerri, Agustín, Arecibo
IGARTUA, SUSANA, Aguadilla
IGARTUA, GREGORIO, Aguadilla
IGUINA, MANUEL A., Río Piedras
Iguina Mora, Martín A., Arecibo
IGUINA REYES, JOSE, A., Ponce
Infante, Rafael, U.A.
Infanzón Ochoteco, Jesús, Hatillo
Iñigo Agostini, Emigdio, Mayagüez
IRIZARRY, JAIME E., Sabana Grande
Irizarry, Sergio, Santurce
IRIZARRY BULLS, BENITO, Arecibo
IRIZARRY BULLS, EDGARDO, Pta. (de Tierra
ISALES, LUIS M., Santurce
ISALES, RAMON, Santurce
Iturrino, José Luis, A.F.

— J —

JANER, FERNANDO H., Río Piedras
JANER, MANUEL, New York
JANER PALACIOS, ANA, Río Piedras

JAVIER DE YOUNG, HAYDEE, Hato (Rey
JESUS, FRANCISCO R. DE, Santurce
JESUS, JOSE A. DE, Caparra Heights
Jiménez, José D., San Juan
JIMENEZ, RAFAEL ANGEL, Caguas
Jiménez Becerra, T., Santurce
JIMENEZ LOPEZ, R., Santurce
Jiménez Mercado, Juan J., Santurce
JIMENEZ PABON, EDWARD, A.F.
JIMENEZ TORRES, CARLOS F., (Ponce
JIMENEZ VELEZ, JOSE L., (Pennsylvania
Johnson, Paul, Humacao
Jordán, Octavio, San Juan
Jouanneau, Luciano, Mayagüez
Juan, Jr., Abel de, Vega Baja
JUAN, ABEL DE, Santurce
JULIA, CARLOS M., San Juan
JULIA, MARIO, Hato Rey
Jung, H. E., Corozal
Justiniano, Raúl T., Bayamón
JUTZY, DONALD ARTHUR, Mayagüez

— K —

KINDY, PAUL E., Mayagüez
KING, ROBERT R., San Juan
KODESCH, J. B., Santurce
KOPPISCH, ENRIQUE, San Juan

— L —

LACOT SALGADO, A., Fajardo
LACOURT, LUIS L., Roosevelt (Hato Rey)
Lafont Zayas, Emilio, Arecibo
Lamela, Manuel A., Cayey
LANDRON, JOSE, Santurce
LANG, ALBERT A., Ponce
LARA, J. J. DE, Ponce
Larrañaga, Jorge, Ponce
LASSISE, ENRIQUE, Sabana Grande
Lastra Morales, O., Canóvanas
LATIMER, JOSE R., Pennsylvania
Latoni, José R., Santurce
Latoni Cabanillas, David, U.A.
LAUGIER, AGUSTIN R., San Juan
Lázaro García, Pedro, Caguas
Lebrón, Domingo, Río Piedras
Lebrón, Francisco A., Santurce
Lee Pimentel, Charles E., Santurce
León Sentenat, Emilio, San Lorenzo
Lehman, Joseph E., Aibonito
Lens, Antonio, Arecibo
Leppink, Harold B., Castañer (Adjuntas)
LERGIER, JULIO E., Santurce
LICHA, JOSE S., Santurce
Lichtenberg, Francisco, Río Piedras
LIMA BEAZ, BERNABE, Río Piedras
LIMERES, JOSE R., E.U.
Lippitt, William F., Santurce
Lizardo, Francis, Río Piedras
LLANO, J. M. del, Santurce
Llavona Vera, Angel F., Ponce
LLOBET, RAMON E., Santurce
Llompart García, J., Santurce
Lluberas, Blanca A., Santurce
Lockhardt, Alfredo, Caguas
Loinaz, Diego, Barranquitas
LOMBARDI, ANTHONY L., Hato Rey
Loperena, Eleuterio, Aguadilla
LOPEZ, HILTON L., Santurce
López, José A., Santurce
López, Waldo E., Santurce
LOPEZ CANDAL, AMAURY, Gurabo
LOPEZ DE LA ROSA, L., Santurce
LOPEZ ELIAS, FRANCISCO, San (Sebastián
LOPEZ GARCIA, RAMON, Caguas
LOPEZ MORALES, HIRAM, Comerío
López Morales, Juan, Fajardo
López Saavedra, Wilfredo, Arecibo
LOPEZ SANABRIA, ULISES, San (Juan
LOPEZ VARGAS, FERNANDO E., (Cidra)
López Vicario, Luis, Ponce
Loyola, Jaime L., Peñuelas
LUGO, ALBERTO E., Río Piedras
Luigi, Hirám, Aguadilla
LUIGUI, J. E., Río Piedras
LUGO, ANIBAL, Santurce
Lugo, Jorge E., Fajardo
Lugo, Orlando, Salinas
LUGO RIGAU, NELSON, Río Piedras
LUM, RALPH J., Santurce
Luzunari, Modesto, Arecibo

— M —

Macossay, Carlos, Río Piedras
Machuca, Fernando, Caguas
Maduro, Luis G., Ponce
MADRAZO, ARTHUR J., Ponce
Maeso, Manuel, Arecibo
MAESTRE, FEDERICO J., Pennsyl- (vania
Magrañer, José A., Adjuntas
MAGRIÑA, RAMON, U.A.

MALAGON, VICTOR, Santurce
MALARET, PEDRO, Bayamón
Malaret, Germán E., New Orleans
MALDONADO, EDUARDO D., (Hato Rey)
Maldonado, Miguel A., Fajardo
MALDONADO, RAFAEL, Santurce
Maldonado Chinea, C., Río Piedras
MALDONADO QUIÑONES, RAMON, (Hato Rey)
MARCHAND, ANGEL M., Santurce
MARCHAND, ERNESTO J., Santurce
MARCHAND, JUAN R., Bayamón
MARCHAND, JUAN S., Manatí
MARCHANY, ANTONIO, Santurce
MARCIAL, VICTOR A., Río Piedras
MARCIAL ROJAS, RAUL A., (Santurce)
MARGARIDA, CARLOS J., Santurce
MARIANI, MIGUEL A., Santurce
Mari Rodríguez, Paul, Santurce
Marín de Rullán, Mimosa, Santurce
MARQUEZ, FRANCISCO A., Aguadilla
MARQUEZ, GUILLERMO S., Arecibo
MARQUEZ, IVAN A., E.U.
MARQUEZ TORRES, J., Aguadilla
MARRERO, HECTOR R., Santurce
Marrero Bonilla, Miguel, U.A.
Márquez Fernández, J., Hato Rey
MARTINEZ, ERNESTO J., Aibonito
MARTINEZ, ERNESTO C., Bayamón
Martínez, Héctor A., Aguirre
Martínez, Hugo, Arecibo
Martínez, Isidro, Arroyo
MARTINEZ, JOSE ANGEL, Arecibo
Martínez, Rosendo, U.A.
Martínez, Zenobio R., Río Piedras
MARTINEZ ALVAREZ, ANTONIO, (Santurce)
Martínez Andújar, R., Lares
Martínez Irizarry, L., Ponce
Martínez Peña, Enrique, Arroyo
Martínez Picó, Amalia, Santurce
MARTINEZ DE POLO, HILDA, (Río Piedras)
MARTINEZ RIVERA, E., Hato Rey
MARTINEZ VILLAFAÑE, HECTOR, (Santurce)
MARTY PEREZ, R., Santurce
MASCORT, JUAN A., Hato Rey
MASSANET, CARLOS L., Río Piedras
Massari Sánchez, F., Morovis
Matos, Angel M., Bayamón
Matta, Carlos, Río Piedras
MATTA, JR., ENRIQUE, Santurce
Matta de Iguina, Fermina, Arecibo
Mattei, Max, Hato Rey
MATTEI, TITO, Yauco
Mattei Vázquez, L., Ponce
Mauleón, Carlos M., Santurce
MAYER, HERBERT, Santurce
Mayol, Miguel A., Morovis
MEDINA, ANTONIO S., Santurce
Medina, Eduardo I., Fajardo
Medina, Gilberto, Humacao
Medina, José T., Santurce
MEJIA CASALS, ALBERTO L., (Santurce)
MEJIA RUIZ, R., Humacao
MEJIAS, FRANCISCO J., Santurce
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Meléndez, Juan, Hato Rey
MELENDEZ, MANUEL, Bayamón
MELENDEZ, PEDRO C., Bayamón
MELLA, JUAN LUIS, Juana Díaz
Menay Jusino, Abelardo, Santurce
Méndez, Arcelio, Utuado
MENDEZ, ARIEL R., Hato Rey
MENDEZ, CEFERINO A., Santurce
Méndez, Jr., Leonardo, U.A.
MENDEZ, NESTOR H., Santurce
Méndez Bryan, R., Santurce
MENDEZ CASHION, DOLORES I., (Santurce)
MENDEZ HERNANDEZ, NICOLAS, (Manatí)
Méndez de Maccosay, L., Río Piedras
Mendín Sabat, José, Santurce
Mendoza Mendoza, Pedro, Aguadilla
Mendoza, Samuel, Bayamón
MENENDEZ CORRADA, R., Río Pie-(dras
Menéndez Mercado, J., Río Piedras
MICSKY, LAJOS VON, Mayagüez
MILAN, ENRIQUE, Santurce
Milán, Ramón, Aguadilla
Milán, Teodoro, Río Piedras
MIMOSO, JUAN, Santurce
Miranda, Ada, Arecibo
MIRANDA, MANUEL N., Río Piedras
Miranda Rosado, Lorenzo, Rincón
MOCZO, LUIS R., Santurce
MOLANO, Pedro A., Río Piedras
Molina, Rafael A., Arecibo
MOLINARI, JOSE G., New York
MONSERRATE, FERNANDO M., (New York)
MONTALVO, ANDRES H., Santurce

MONTALVO, JOSE H., Añasco
Montalvo, Julio R., Añasco
MONTALVO, LUIS J., Santurce
MONTALVO DURAND, E. A., (Río Piedras)
MONTALVO GUENARD, A., Santurce
Montes, Alberto L., Vega Baja
Montes Esteves, Santiago, Moca
MONTILLA, EDUARDO, Santurce
MONTILLA, VICTOR J., Santurce
Mora Boneta, Pedro, Arecibo
MOORE, CHARLES B., Mayagüez
MOORHEAD, JOHN S., St. Thomas, (V.I.)
MORA DE NOCHERA, MARIA T., (Mayagüez)
MORALES, CARLOS E., Río Piedras
MORALES, JUAN E., Río Piedras
MORALES, LUIS A., Ponce
MORALES, LUIS M., Puerta de Tierra
MORALES, PABLO LUIS, Santurce
MORALES OTERO, PABLO, Santurce
MORALES BOYER, RAFAEL, (Río Piedras)
MORALES MUÑOZ, M., Santurce
MORALES RUIZ, J. S., Bayamón
MORENO, NARCISO, Santurce
Moreta Moreno, René, Guaynabo
Morillo, Luis, Peñuelas
Morris, Oriel C., Mayagüez
Moscoso Cordero, J. M., Cayey
MUDAFORT, RAFAEL A., Manatí
MUDAFORT, SARKIS M., Santurce
Mulero, Héctor L., Santurce
MUNDO FRANCISCO E., Santurce
MUÑIZ, RADAMES, Hato Rey
Muñiz, Antonio, San Sebastián
Muñoz, AUREA, Hato Rey
MUÑOZ, JUSTO LUIS, Villalba
Muñoz Candelario, A.L., Arecibo
MUÑOZ DONES, ELOISA, Río Piedras
MUÑOZ MacCORMICK, C. E., Santurce
MUÑOZ VAZQUEZ, RAFAEL, Juana (Díaz
MURPHY, IAN D., Santurce
MUSSENDEN, BORINQUEN, E.U.
McCANDLES, JAMES, San Sebastián
McCONNIE, RANDOLPH J., Santurce
Macpherson, Roderick, Arecibo

— N —

NADAL, HECTOR M., Santurce
NADAL GRAU, J., Mayagüez
Najul Bez, E., Isabela
NAVARRO, FELIX A., E.U.
Navarro, Francisco, Aguadilla
NAVAS, ANTONIO, Santurce
NAZARIO RODRIGUEZ, R., California
NEGRETE, CESAR A., Cabo Rojo
Nero Viera, Pedro del, Caguas
Nido Stella, Roque, Bayamón
Nieves, Luis Angel, Río Piedras
Nieves, Lydia D., Fajardo
Nieves Calcaño, Gilberto, Fajardo
NIEVES COLON, J., Santurce
Nieves López, Pedro L., Río Piedras
NINE CURT, JOSE, Humacao
NOCHERA, DOMINGO, Mayagüez
Noguera, Juan A., Santurce
NOGUERAS, JUAN J., Hato Rey
Nogueras, Julio, U.A.
NOVOA CABALLERO, MIGUEL, (Caguas)
NOYA BENITEZ, JOSE, Santurce
NUÑEZ COLON, NEIDA, Cataño
Núñez Santana, Idalia, Santurce

— O —

Ocasio Cabañas, K., Puerto Nuevo
OLIVELLA, JAIME A., Río Piedras
OLIVERAS GUERRA, A., Santurce
Oliver Pino, Gabriel, Yauco
OLMEDO, JOSE C., Maryland (A.F.)
O'NEILL, EDWARD, Santurce
Orlandi, Radamés, Santurce
ORPI, JR., PEDRO, Santurce
ORTEGA, LUIS, San Juan
Ortega, F. Armando, Coamo
Ortega, Ramón C., Río Piedras
ORTIZ, ANTONIO, Santurce
Ortiz, Julio A., Humacao
ORTIZ, YDALIA, Bayamón
ORTIZ GORDILLS, EDGARDO, A.F.
Ortiz Guzmán, Guillermo, Puerta de (Tierra
Ortiz Santiago, Pedro M., Toa Alta
Otero, Pedro J., San Juan
Otero Colón, Angel M., Bayamón
OTERO LOPEZ, A., Manatí
OTERO ROQUE, MANUEL, Santurce

— P —

Pablos, Glicelia, Arecibo
Pacheco, Luis A., Caparra Heights
PADILLA, ANGEL F., Cabo Rojo
Padrón, Jorge, San Sebastián
Pagán, J. E., Arecibo
Pagán, Luis H., Santurce
PADRO, FERNANDO, Puerta de Tie- (rra
Pagán, Rafael Angel, Río Piedras

Pagán, Victoriano, Bayamón
Pagán, Víctor J., Caparra Terrace
Pagán, Víctor M., San Sebastián
Pagán Carlo, J., Arecibo
Pagán Pascual, Víctor M., Bayamón
Palacios, Miguel A., Mayagüez
PALMER LOPEZ, ARNALDO, (San Juan)
PALMIERI, JULIO, Santurce
PALOU, PEDRO J., Humacao
PANIAGUA, MANUEL E., San Juan
Paravisini, F., Jayuya
Pardo, Miguel A., Santurce
PARES DAVILA, MARIA A., Santurce
Parodi, Francisco, Ponce
Parrilla, Eduardo, Arecibo
PASSALACQUA, JOSE R., Santurce
PASSALACQUA, LUIS A., Santurce
Pastor, César A., Ponce
Patiño, Bolívar, Bayamón
PAVIA FERNANDEZ, MANUEL, (Santurce)
Peguero Medina, Julio A., Santa Isabel
PELEGRINA SARIEGO, IVAN, (Santurce)
Pellerano, Arturo, Yabucoa
Pendleton, Adeline, Humacao
PEÑA JOSE A., Cayey
Peralta, Luis E., Guánica
PEREA, AUGUSTO, Mayagüez
PEREA, LUIS R., Mayagüez
PEREA, NELSON, Mayagüez
Pérez, Consuelo, Luquillo
PEREZ, EDUARDO R., Arroyo
Pérez, Hilton, U.A.
Pérez Arzola, Miguel, Santurce
PEREZ GONZALEZ, SANTIAGO, (Barceloneta)
PEREZ GUARDIOLA, LYDIA, (Santurce)
PEREZ LARA, RAFAEL, Georgia (A.F.)
PEREZ MARCHAND, DOLORES, (Ponce)
Pérez Montalvo, J., Arecibo
Pérez Matos, J. A. Utuado
PEREZ RIBIE, RAMBERTO, Ponce
Pérez Roig, Manuel, U.A.
PEREZ SANTIAGO, ENRIQUE, (San Juan)
PEREZ TOLEDO, A., Santurce
Pérez Roig, Manuel, Ponce
PICO, GUILLERMO, Santurce
PICO, JOSE, Santurce
Picó, José F., Bayamón
Pietri, Agustín, E.U.
Piñero, B., Aguadilla
PIÑERO, DOLORES M., Santurce
Piñeyro, J. Ramón, Carolina
Piñeiro, Ramón, Fajardo
Piñero Fernández, R., Río Piedras
Piñot Nin, Ramón A., Santurce
Pintado, Isidoro, Moca
Piovanetti, Simón, Hato Rey
PLA, JOSE S., Santurce
Polanco, Gerardo, Bayamón
Polanco González, José, Caguas
Polanco Rivera, José E., Caguas
PONS, JR., EDUARDO R., Santurce
PONS, JUAN A., Santurce
PORRATA, FRANCISCO C., Ponce
PORRATA, JOSE LUIS, Hato Rey
PORRATA DORIA, R., Santurce
Portela, Angel, A.F.
Portela, Ismael, Arecibo
Pou, Angel E., Arroyo
POU, JAIME F., Hato Rey
PRADO, JOSE del, Ponce
PRIETO, EZEQUIEL, Caguas
Pronko, Robert, Humacao
Pujadas, Juan S., Bayamón
PUJADAS DIAZ, MANUEL, Santurce

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QUEVEDO BAEZ, MANUEL, Santurce
Quetglas, Miguel, Hato Rey
QUILICHINI, CARLOS A., Ponce
Quinquilla, Rafael A., Hormigueros
Quintana, Manuel A., Luquillo
QUINTERO, ERNESTO, Santurce
QUIÑONES, EDGARDO, San Germán
QUIÑONES, JESUS M., Santurce
QUIÑONES, NORBERTO A., Santurce
Quiñones, Víctor M., Santurce
QUIÑONES ACOSTA, F., Santurce
QUIÑONES JIMENEZ, FRANK, (Santurce)
QUIÑONES JIMENEZ, NICOLAS, (Santurce)
QUIÑONES JIMENEZ, JR., N., (Santurce)

— R —

RABELL, GUALBERTO, Santurce
RAFFUCCI, F. L., Santurce
Ramírez, Dixon, Cabo Rojo
RAMIREZ, ELI A., Hato Rey
RAMIREZ, KENNETH, Aguadilla
RAMIREZ, RAMON E., Mayagüez
Ramírez, Ricardo, Fajardo

RAMIREZ, URBANO, Corozal
Ramírez, Víctor, Cayey
Ramírez de Arellano, G., San Juan
RAMIREZ DE ARELLANO, MAX, (Santurce)
Ramírez Cuerda, T., Mayagüez
RAMIREZ KOHL, EMILIO, Roosevelt (Hato Rey)
RAMIREZ LEDESMA, JAFETH, (Mayagüez)
RAMIREZ MARINI ARQUELIO, (San Germán)
Ramírez Santisteban, G., San Juan
RAMIREZ SANTOS, R., Río Piedras
Ramírez Silva, Amador, Santurce
RAMIREZ WEISER, R., Bayamón
Ramos, Raoul L., Missouri
RAMOS CASELLAS, P., Santurce
RAMOS ELVIRA, E., Cayey
Ramos Gómez, Jacobo, Bayamón
RAMOS GONZALEZ, LUIS, Navy
RAMOS ISERN, FRANCISCO, Caguas
RAMOS LEBRON, JOSE, Fajardo
RAMOS MIMOSO, JOSE R., Bayamón
RAMOS OLLER, A., Santurce
Ramos Yordán, F., Aibonito
Ramos Yordán, Juan H., Ciales
Ramos Yordán, Luis E., Hato Rey
Raymond, Félix A., Loíza
Reads, Francisco, Las Marías
Read Medina, Pedro Julio, Lajas
Rechany, J. E., Santurce
REICHARD, WILLIAM, Santurce
REMUS, LUIS E., Morovis
RENGEL, RICARDO E., A.F.
Reyes, Antonio R., Hato Rey
REYES, FELIX M., San Juan
Reyes, José Manuel, Las Piedras
Reyes, Juan J. Félix, Bayamón
Reyes Matos, Félix, Fajardo
REINHARDT, ALICE V., Río Piedras
RICE-WRAY, EDRIS, Río Piedras
Richards, Lawrence, Arroyo
RIGAL RIERA, RENE, Ponce
Riera López, Salvador, Santurce
RIFKINSON, N., San Juan
RIGAU, GABRIEL, Río Piedras
RIGAU MARQUES, JOSE M., (Santurce)
RINCON, VICTOR, Humacao
RIO, JUAN DEL, Bayamón
Ríos, Manuel J., Aguadilla
Ríos, Ramón A., Santurce
RIOS PAGAN, J.A., Quebradillas
Rivas Marín, H. M., Santurce
Rivera, Dennis, Salinas
Rivera, Eva, Caguas
Rivera, Gerant, Río Piedras
RIVERA, GILBERTO, Río Piedras
RIVERA, JULIO V., Río Piedras
RIVERA, KATHERINE, Río Piedras
Rivera, José H., San Juan
RIVERA, RAFAEL, Caguas
RIVERA, RAUL R., Santurce
RIVERA, VICTOR M., Santurce
Rivera Aromí, V., Ponce
RIVERA ASENCIO, VICTOR M., Río (Piedras
RIVERA AULET, R., Arecibo
Rivera Ayala, G. A., Santurce
RIVERA BIASCOECHEA, Z. A., (Arecibo)
RIVERA BRUNO, HECTOR J., A.F.
Rivera Cestero, Francisco, Caguas
RIVERA CINTRON, FRANCISCO, (San Juan)
Rivera Díaz, Jorge, Ponce
RIVERA LUGO, CARLOS, Santurce
RIVERA LUGO, ILDEFONSO, (Río Piedras)
Rivera Muñiz, Víctor M., Bayamón
Rivera Olivieri, Félix M., Arecibo
Rivera Otero, Antonio, Bayamón
RIVERA OTERO, J. M., Santurce
Rivera Pratts, J. E., San Sebastián
RIVERA PORRATA, P. A., Santurce
Rivera Rodríguez, Freya, Vega Baja
Rivera Rodríguez, Rodolfo, Arecibo
RIVERA ROMERO, ENRIQUE, Juncos
Rivera Torres, Pedro, Caguas
RIVERA TRUJILLO, ANTONIO, (San Juan)
ROBERT, JOSE L., Santurce
ROBERT DE RAMIREZ, MARIA I., (Santurce)
ROBERT DE ROMEU, MARTA, (Santurce)
Robles Castro, Elí, Fajardo
ROCA, JULIO C., Yauco
RODRIGUEZ, ARMENIA, Puerto (Nuevo
RODRIGUEZ, CALIXTO, Ponce
RODRIGUEZ, EDITH Z., Santurce
RODRIGUEZ, ELVIRA, Ponce
Rodríguez, Gilberto L., Ponce
RODRIGUEZ, HECTOR F., Ponce
RODRIGUEZ, JUAN C., Bayamón
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ASAMBLEA ANUAL

Diciembre 6-10, 1955

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BOLETIN DE LA ASOCIACION MEDICA DE PUERTO RICO, published monthly at San Juan, Puerto Rico, for ----------------------, 19------.

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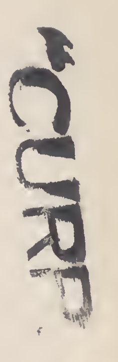
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DE LA

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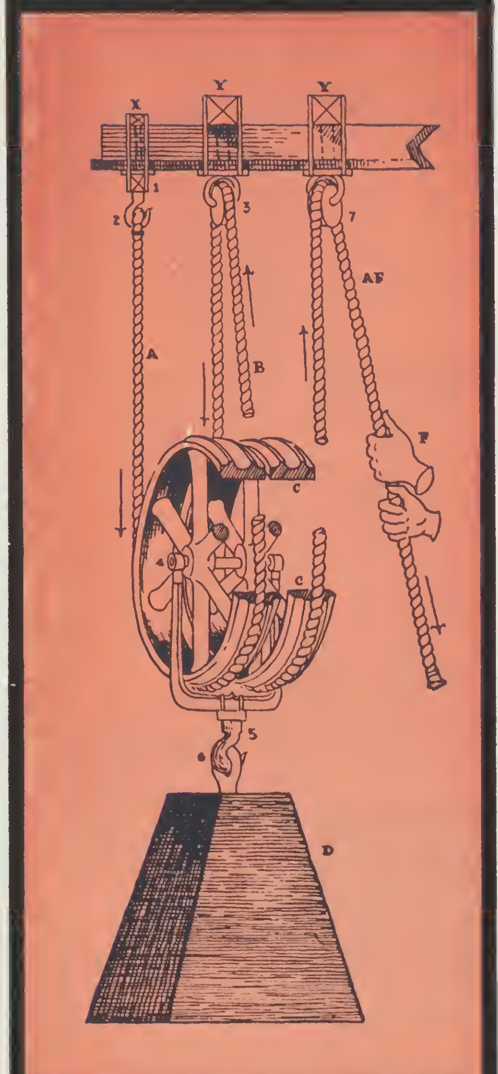
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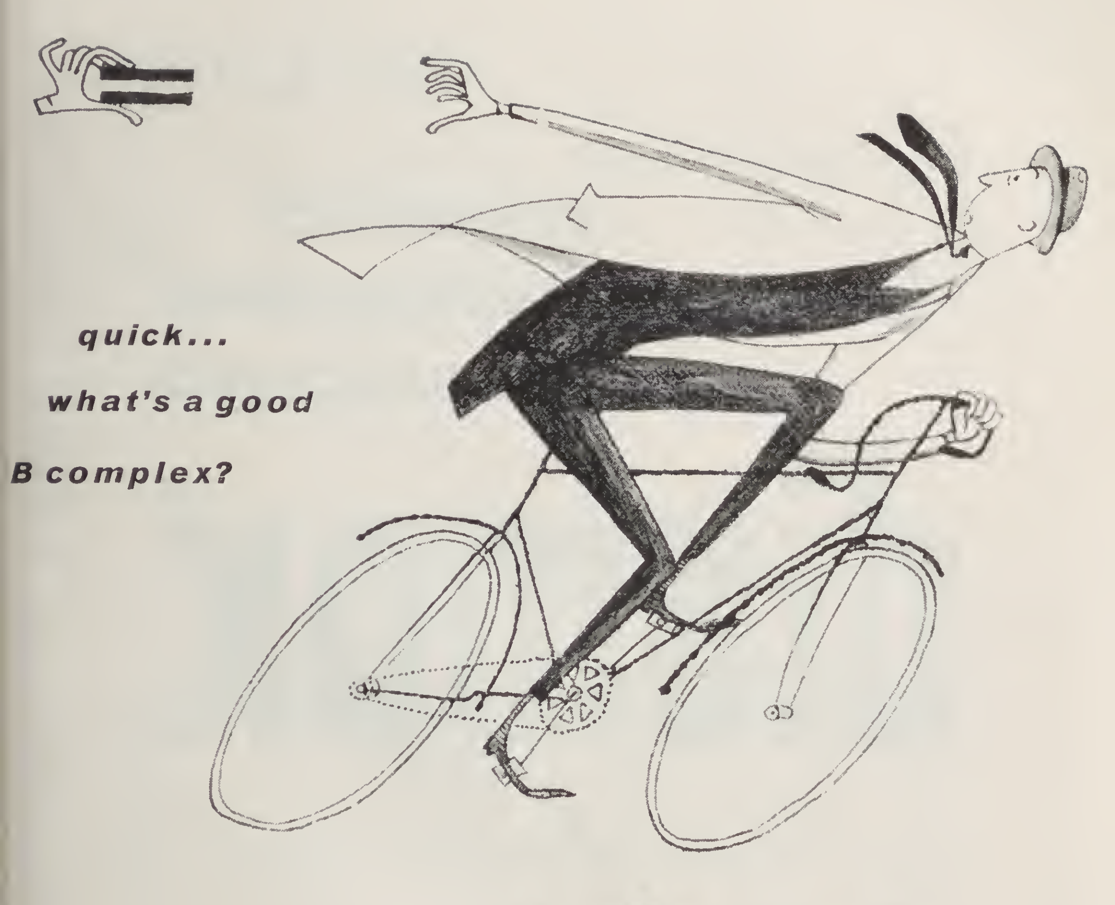
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DE LA ASOCIACION MEDICA DE PUERTO RICO

VOL. 47 NOVIEMBRE, 1955 No. 11

SCHISTOSOMIASIS MANSONI

THE ACUTE PHASE

REPORT OF A CASE

JENARO HADDOCK-SUAREZ, M.D.
and
RAFAEL RODRIGUEZ-MOLINA, M.D., Sc.D.

A white male, age 24, Korean Veteran, admitted to PM&R Service of San Patricio Hospital on December 9, 1954 because of history of pain and stiffness in the neck of sudden onset accompanied by fever and chills of four days duration. Anorexia, headaches, pain in lower extremities and dry cough had been present also. Chills and fever had occurred daily since onset of illness, the fever being followed by profuse sweating. Cough had not been associated with chest pain. Patient returned from Korea the month previously. No history of malaria while there. He bathed frequently in rivers while in Korea, but not in Puerto Rico since childhood. No history of drinking raw milk or exposure to rats.

Physical examination revealed a young male appearing acutely ill with flushed fascies. Blood pressure 110/80, pulse 80, no icterus or conjunctivitis. Liver edge was felt 1½ fingerbreath below the right costal margin. Tip of spleen was just felt at costal margin. The initial impression made by Physical Medicine and Rehabilitation Service was acute myositis of cervical and paravertebral muscles, but when the white blood count was reported as being 26,000 with 59% Eosinophils, the patient was immediately transferred to the Medical Service.

Laboratory findings: Several smears for malaria taken at height of fever and during chills were reported negative. Urine showed some hyaline and granular casts, traces of albumin and specific gravity of 1.026. It became negative a few days after admission. Spinal fluid revealed seven cells per cmm with 40%

* Veterans Administration Center, San Patricio Hospital, San Juan, Puerto Rico.

polys and 60% lymphocytes. Normal amounts of protein, chloride and glucose. Spinal fluid culture was negative after 48 hours. Blood culture made on admission showed no growth after 10 days. Blood serology - negative. Non protein nitrogen - 29 mg.%. On admission, 12/10/54, RBC - 4,800,000 - Hgb 15.0 gm; 2/8/55 - RBC 4,880,000 - Hgb 15.0gm. On 1/10/55 - Sedimention rate - 10.0 mm. after 60 minutes. Volume of Packed RBC - 50 mm.

Dilutions

Agglutinations	1:20	1:40	1:80	1:160	1:320
Typhoid "O" antigen	4	3	2	1	—
"H" antigen	2	2	2	2	1
Paratyphoid A	1	+	—	—	—
B	1	1	+	—	—

Agglutination with B Abortus antigen - negative. Routine and Brucella blood cultures were reported negative 21 days after innoculation under CO_2.

Liver function tests

12/13/54—Serum bilirubin 0.18 mg.%. Cephalin flocculation: 24 hrs. — 2 +; 48 hours — 3 +. Prothrombin time — patient 14.0 sec. Control 11.0 sec.

1/10/55 —Cephalin flocculation — 24 hrs. — 3 +; 48 hours — 4+.

1/11/55 —Prothrombin — Patient — 11.4 sec. Control 11 sec.

1/24/55 —Thymol turbidity — 10.5 units.

1/24/55 —Bromsulfalein 8% after 45 minutes. Cephalin flocculation: 24 hours — 3 +; 48 hours — 4 +.

2/8/55 —Bromsulfalein — 12%. Cephalin flocculation: 24 hrs. — 2 +; 48 hours — 3 +.

Other Blood Chemistry Studies

12/13/54—Total protein 8.3 mg%.
Albumin 3.7 — Globulin 4.6.

1/10/55 —Total protein 8.0 mg%.
Albumin 4.0 — Globulin 4.0.

2/8/55 —Total proteins 6.6 mg%.
Albumin 3.7 — Globulin 2.9.

2/14/55 —Alkaline phosphatase — 11.0 units (Bodansky).

1/24/55 —Sedimentation rate — 4.0 mm after 60 minutes.

Tests for Schistosomiasis

2/10/55 —Skin tests with cercarial and adult worm antigen were strongly positive.

*2/12/55—Circumoval precipitation test was also positive. Complement fixation employing cercarial and adult worm antigens was positive (4+) in 1:12 dilution.

Course in the Hospital: During the first week of hospitalization there was not much change in the clinical picture. Patient continued to complain of pain and stiffness of neck and moderate dry cough. Spleen became definitely palpable. Spiking temperature continued. Diagnosis remained obscure but possibilities considered were collagen disease or larval or acute phase of a parasitic disease. Spinal puncture was repeated with normal findings. No pathologic reflexes were elicited.

On December 20, and while afebrile, patient suddenly developed generalized pruritic rash over skin of abdomen, back of chest and legs. This rash resembled scabies. However, scrampings from various lesions were negative for the mite. The condition was not definitely diagnosed scabies by dermatology consultant and he also believed it could be an "id" reaction to the parasite. On this day patient admitted to have been fishing for fresh water shrimps in a brook near the town of Aibonito, which is a well known endemic center of Schistosomiasis. The last time he went fishing was about the middle of November, but does not recall the exact date. He had fished and swum in the same river several times previously. Blood smears for L. E. cells were negative on December 23, 1954.

By the tenth hospital day patient was afebrile and felt much improved from constitutional symptoms and was granted a pass on December 23. On December 28, a sigmoidoscopic examination was performed and rectal mucosa was reported of normal appearance. Rectal biopsy was done and found negative for Schistosoma ova. On December 29, patient was given another pass. He returned on January 1/55 stating that on previous day, that is the following day after leaving the hospital, he had had a severe chill accompanied by fever and profuse sweating. The liver was now palpable 3 fingerbreadth below the costal margin and it was tender. Spleen was just palpable. At this point the ward physician in charge of the case felt that Collagen disease or Lymphoma were the most likely possibilities, as the fever suggested the Pels-Epstein type. A liver biopsy was performed January 4, 1955. The pathologist's

* These tests were performed through the courtesy of the Department of Parasitology, School of Medicine, and the Army Tropical Research Medical Laboratory.

report reads as follows: "A definite specific histologic diagnosis in this case is not possible. The lesion in the liver is a non-specific granuloma with an eosinophilic infiltrate. An early acute schistosomiasis granuloma of liver can not be ruled out, nor a reaction of the reticulo-endothelial system to some unknown stimulus."

The patient continued febrile during the next three weeks. He received treatment for ascaris and hookworm infections. He had lost 19 pounds since admission. On January 12, for the first time, stools were reported positive for Schistosoma ova. On January 7, patient complained to nurse that for about two days he had had four or five semi-solid bowel movements, soon after eating and accompanied by generalized abdominal distress. On January 12 he passed several liquid stools with mucous and fresh blood. Another sigmoidoscopic examination was performed January 13, 1955 and the instrument was introduced only five inches, limited by pain and discomfort. The entire mucosa was red, granular, friable, bleeding easily, an appearance very suggestive of schistosoma mansoni infection. No ulcerations were seen. However, no schistosoma ova were observed in the specimen removed from a rectal valve. At this time further specimens of stool were persistently positive for schistosoma mansoni ova. Another sigmoidoscopic examination performed January 25 revealed an inflammed mucosa infiltrated with hemorrhagic areas. Biopsy of tissue was positive for ova. By the end of January, patient was again afebrile and presented no complaints and was gaining weight. Eosinophilic leucocytosis was still present, however. As this report is written, 3/16/55, patient has regained lost weight and has remained afebrile, but eosinophilia persists. A second liver biopsy was performed January 19, 1955 with the following report: "Non-specific granuloma of liver, probably due to schistosoma mansoni". Treatment for Schistosomiasis was begun 2/14/55. Sodium antimonyl gluconate (Triestam) was employed.

COMMENT ON CASE

The term Bilharzial fever as employed in the literature on diseases of the tropics is defined as a toxic or allergic state appearing between the 4th and 6th week after bilharzial infestation and characterized by fever, general symptoms of ill-health, urticaria and eosinophilia. A recent schistosomal infestation may reveal itself clinically by a fairly severe illness as observed in our case, characterized by pyrexia, cough, hepato-splenomegaly, urticaria and eosinophilia. The case just presented, exhibited all these manifestations except the urticaria. This acute and characteristic illness has been described in Puerto Rico[1,2] and elsewhere but it is rarely observed by the practicing physician. In the majority of

cases these toxic or anaphylactoid reactions may be mild and the clinical picture may amount to merely disturbance of the general health. Loss of weight, lack of appetite, headache, abdominal and limb pains, slight pyrexial attacks, with possible transient urticarial eruption and hepatosplenomegaly may then be the only indications of a possible bilharzial infection. In Puerto Rico even this type of milder reaction is quite often either not observed by a physician or is diagnosed as being something else.

The temperature, eosinophilia and cough are believed to be due to the intravascular migration of the young flukes through the lungs and other organs of the body. Later when the worms have attained maturity and copulation and oviposition have taken place in the hemorrhoidal plexus of veins, signs of localization of the disease in the colon are evident as manifested by diarrhea, mucous and blood in the stools. These features were quite evident in our present case.

Fairley,[3] in discussing the pathological changes produced in the acute or toxemic stage of schistosomiasis, mentions that the anaphylactoid reaction in the sensitized tissues of the host plays an important part in the production of both pathological lesions and the clinical picture. He classified the toxic substances as (1) specific glandular secretions produced by the cercariae, schistosomes and miracidia containing enzymes; (2) byproducts of metabolism; and (3) excretions to include the katabolites of hemoglobin, such as bilharzial pigment.

Miyagawa and Takemoto[4] conducted experiments on mice which had been immersed in water previously infested with carcariae, which is the infective stage of the worm. As a result of these studies they found that the main route of invasion of Schistosoma japonicum from the skin to the portal veins of the host was as follows:

"The young worms penetrate actively into the skin, principally into the lymphatic spaces, then for the most part they invade the blood capillaries or the small peripheral veins, later gathering in the right side of the heart. Some of those in the skin tissue pass by the lymphatic vessels to the lymphatic glands, in which some are arrested and killed; others, however, being able to pass through into the emergent veins finally reach the right side of the heart. The worms in the right side of the heart now pass to the lungs. They are stopped for short time in the lung tissue, owing to the disproportion in the size of the worms and alveolar capillaries. Finally, they return to the left side of the heart and are carried through the aorta to the wall of the gastro-intestinal canal, after passing through which they reach their per-

manent residence (the portal branches in the liver) by the mesenteric veins."

It may also be deduced from their experiments that the schistosomula pass to the lungs from the skin at varying rates from 2 - 5 days, and after a slight hold-up in the lung tissue they begin to reach the liver from 3 days onwards. In one of their experiments the liver of an infected mouse 5 days after initial infection showed numerous schistosomula.

In the liver the male and female schistosomes live separately, but about 4 weeks after entering the body they become sufficiently mature to enter their period of sexual activity, join in pairs, and travel into the mesenteric veins forcing their way against the blood stream towards the veins of the submucosa of the bladder or large intestine. At this stage of migration it is probable that toxic and antigenic material is liberated into the general circulation. According to Girges[5] this journey coincides with the acute or toxaemic stage. Clinical studies have shown that this stage usually comes on 4 - 6 weeks after initial exposure. At this stage the bilharzial complement fixation test becomes positive, eosinophilia appears and the anaphylactoid reaction, including urticaria, pyrexia and pulmonary infiltration, may appear. The underlying cause of this allergic reaction is unknown, but it is probable that the body becomes sensitized to one or more of the schistosome toxic substances described by Fairley.[3]

Special attention should be drawn to the pulmonary changes in bilharzial fever.

It is often stated that these are due to the impaction or migration through the lungs of developing schistosomula. It is however known that a similar state is found in other parasitic infections which do not include a stage of pulmonary migration. Hence lung infiltrations have been described in cutaneous helminthiasis, amoebiasis and trichiniasis. Furthermore, extracts of ascaris and even of histamine injected into guinea pigs have been shown to produce an identical pathological picture.[5] We have no x-ray evidence of pulmonary involvement probably because we did not look for it.

Accordingly all the features of bilharzial fever can best be represented as an anaphylactoid reaction towards certain toxic substances produced by mature schistosomes at the stage of migration from the liver to the final end organs.

SUMMARY

1. A case of Schistosomiasis mansoni in a white Puerto Rican male, age 24, and believed to be suffering from acute or toxic phase of the disease has been presented.

2. The illness characterized by sudden onset of chills and fever, accompanied by pain and stiffness of the neck, headache, pain in the lower extremities, dry cough and anorexia. There was history of river bathing and fishing about three weeks prior to onset of symptoms.

3. The diagnosis of the condition was not definitely suspected until report of liver biopsy performed about one month after onset of illness was received and a non-specific granuloma with eosinophilic infiltrate was observed. Schistosomiasis mansoni was then suspected.

4. On January 12, slightly over a month after admission to hospital, the stools were first found to be positive for schistosoma mansoni ova. On this date sigmoidoscopic examination revealed an inflammed, red, granular rectal and colonic mucosa suggestive of schistosomiasis. One week previously patient had diarrhea, with mucous and blood in stools.

5. A marked and persistent eosinophylic leucocytosis has been observed during the clinical course which included periods of pyrexia and apyrexia. The eosinophilia has persisted, even though the patient remains symptom free.

6. Intracutaneous reaction to cercarial and adult worm antigen extracts were markedly positive. The complement fixation reaction was markedly positive. Ault and cercarial antigens were employed. The circumoval precipitin reaction demostrative of specificity for Schistosoma mansoni was positive also.

7. Liver biopsies do not indicate fibrotic or cirrhotic changes.

8. Liver function studies indicative early liver damage.

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TABLE I

SCHISTOSOMIASIS MANSONI
Acute Phase
Stool and Rectal Mucosa Examination

Patient admitted: December 9, 1954

Date	Stool	Mucosa	Positive for:
12/13/54	x		Ascaris; Hookworm; and Trichuris ova.
12/14/54	x		Same as above
12/15/54	x		Same as above
12/27/54		x	Negative for Schistosoma ova
1/3/55	x		Ascaris; Hookworm; and Trichuris
1/12/55	x		**Direct smear**: Schistosoma ova: Alive-7; Dead-12
1/13/55		x	Negative for Schistosoma ova
1/13/55	x		Schistosoma ova: Alive-0; Dead-10
1/14/55	x		Schistosoma ova: Alive-2; Dead-8
1/17/55	x		Schistosoma ova: Alive-3; Dead-9
1/18/55	x		Schistosoma ova: Alive-0; Dead-6
1/20/55	x		Schistosoma ova: Alive-2; Dead-3
1/24/55	x		Schistosoma ova: Alive-1; Dead-7
1/25/55		x	Schistosoma ova: Dead collapsed-2
2/14/55	x		Hookworm; Trichuris
2/15/55	x		Trichuris

TABLE II

Acute Phase

Total and Differential Counts

Patient admitted: December 9, 1954

DATE	White Blood Cells Thousands per cu. mm.	Polymorpho-nuclear Neutrophils %	Eosinophils %	Lymphocytes %	Monocytes %	Other Cells %
12/10/54	26,450	24	59	17	0	0
12/13/54	30,000	17	80	3	0	0
12/15/54	27,000	13	74	8	0	0
12/17/54	26,100	14	75	11	0	Atypical-5% Lymphocytes
12/28/54	20,500	19	60	21	0	0
1/3/55	10,700	62	20	18	0	0
1/10/55	27,650	12	77	11	0	0
1/24/55	31,950	16	80	4	0	0
2/8/55	15,650	36	40	24	0	0
2/14/55	21,000	36	40	24	0	0
2/15/55	13,500	27	52	17	0	0

FEMALE PSEUDOHERMAPHRODITISM OF ADRENO-CORTICAL ORIGIN:

REPORT OF A CASE AND DISCUSSION OF CURRENT CONCEPTS OF PATHOGENESIS AND TREATMENT

*MANUEL E. PANIAGUA, M.D.**

Abnormal genital development including several degrees of intersexuality has been a subject of great interest on the part of scientists and laymen alike since remote antiquity. However, it has not been possible until relatively recent times to distinguish the aberrations of gonadal development which lead to true hermaphroditism from the endocrine dysfunctions which give to a genetically and anatomically complete individual the external appearance of the opposite sex. Notable among the latter is the so-called adreno-genital syndrome, a virilizing syndrome which causes pseudohermaphroditism in the female and precocious genital and somatic development in the male.

Probably a congenital disturbance, it is usually manifested at birth or shortly thereafter, although some patients do not develop the full-blown syndrome until puberty. When virilization appears in the adult it is usually accompanied by disturbances of carbohydrate and electrolyte metabolism characteristic of Cushing's syndrome.

Until the past few years these cases were scientific curiosities of academic interest only, since not much could be done for them. With the demonstration by Sprague and co-workers[1] that cortisone administration reduced the excretion of 17-ketosteroids in normal men, and the application of these findings by Wilkins[2] in the adrenogenital syndrome, whole new vistas have opened for the scientific study of this disturbance and its possible treatment.

We are concerned in the present communication with the metabolic studies and the results of both medical and surgical treatment which have entirely changed the life of one of these unfortunates.

Case Report:

E.F.M., a 15-year-old colored girl, was first seen in our OPD because of "amenorrhea" of four months duration. A more detailed history revealed that she had had a slight vaginal bleeding some four months before (which was interpreted as a first menses)

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and had not bled since. She claimed she had had a rather low-pitched, deep voice and moderate hirsutism since the age of 8 or 9.

The past and family histories were non-contributory.

The phyesical examination revealed a short, stocky, muscular colored girl with a deep voice. There was a thin "moustache" on the upper lip and abundant hair on the face and throughout the body. The distribution of the pubic hair was of the male type. The breasts were flat with very little if any breast tissue. The external genitalia (Fig. 1) showed a phallus about 4-5 cm. long with a well formed glans and prepuce but no urethral opening. The prepuce was continuous with the labia minora. The urethral opening was located below the base of the phallus and a little farther down there was a very small vaginal opening which could not be explored in its entirety. Rectal examination failed to reveal the presence of any pelvic organs.

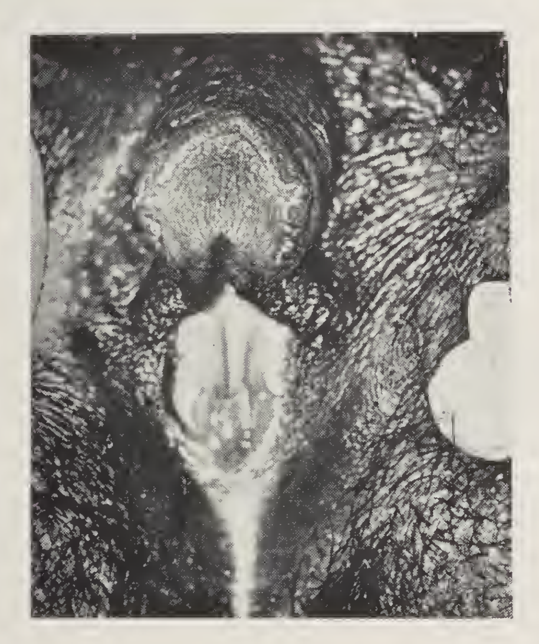


Figure 1

A tentative diagnosis of adrenogenital syndrome with pseudohermaphroditism was made and the patient was hospitalized for observation and treatment.

While in the hospital, the following laboratory studies were performed with the accompanying results: RBC - 4.56 million; Hgb. - 14.3 gm. (98%), WBC - 9,500 with a normal differential count. Urinalysis essentially normal. Stool - T. trichura. 17-ketosteroids - 50.44 mg. in 24 hours. Eosinophile response to a single i.m. injection of 24 units of ACTH - 75% decrease in 4 hours.

Kepler test showed normal values. Glucose tolerance test showed a rather flat curve with a maximum elevation of 28 mg. % above the fasting level at one half and one hour after ingestion of glucose. BMR - +4%. Blood chlorides 580 mg.%.

Radiological study of the skeleton was reported as showing "accelerated development". Tomography of the abdomen after presacral air insuflation revealed some apparent enlargement of the left adrenal gland and a questionable soft tissue shadow partially superimposed on those of the left kidney and spleen.

With the additional information obtained from these studies it was decided to give the patient hydrocortisone. She was started on 40 mg. every six hours and 1 mg. of stilbestrol was added to the regime. After four days hydrocortisone was reduced to 100 mg. daily and this dosage was continued for a whole month, when it was further reduced to 80 mg. daily.

After two weeks of treatment her 17-ketosteroids were down to 25.65 mg. in 24 hours, and three weeks later, to 16.79 mg. in 24 hours. Daily eosinophile counts performed throughout the period of treatment varied between 25 and 200. Clinically, the patient improved considerably, losing most of her excess hair, gaining weight and developing more feminine body contours.

During all this time she had been on a low sodium diet without potassium supplementation. About five weeks after the beginning of therapy, in the middle of the night, she had an episode of weakness accompanied by a transient elevation of her blood pressure. An ECG failed to reveal any abnormality but she responded rapidly to the intravenous administration of potassium chloride with complete disappearance of symptoms. From then on oral KCL was added to her regime.

Dr. Jenaro Suárez, who managed the patient jointly with us, devised a method of progressive dilatation of the hypoplastic vagina by means of insertion of test-tubes of gradually increasing size. Amputation of the phallus and plastic reconstruction was performed by him after the patient had reached the maximum degree of improvement.

In the meantime her 17-ketosteroids excretion had continued in the range of 16-18 mg. in 24 hours, during her second month of therapy. Hydrocortisone was discontinued in order to observe the duration of its suppressing effects, but two weeks later her 17 ketosteroids were back up to 44 mg. in 24 hours, while her eosinophiles ranged from 150 to 300. Re-institution of therapy caused a decrease of 17-ketosteroids to 17.9 mg. in 24 hours within two weeks.

Since it was economically impossible for this patient to continue indefinitely on hydrocortisone, a consultation was held with

Drs. Jenaro Suárez and José Noya-Benítez on the advisability of doing a subtotal adrenalectomy. It was decided, after much debate, to use an abdominal approach for exploration and adrenalectomy in one stage. The operation was performed by Dr. Noya-Benítez assisted by Drs. Pedro A. Suau and Jenaro Suárez, and exploration of pelvic organs was carried out at the same time. The uterus was hypoplastic but the ovaries and tubes looked normal. The left adrenal and one half of the right one were removed and a biopsy of the right ovary taken. The pathologic study revealed multiple extracortical nodules (which are found in 40% of adrenals). The ovarian tissue was normal.

The patient had an uneventful recovery accompanied by further clinical improvement in the form of complete loss of facial hair, marked reduction in body hair and gradual change of pubic hair to a more feminine distribution. Her skin became softer and she almost "shed" her former epidermis.

Her 17-ketosteroids remained in the range of 23-26 mg. in 24 hours in spite of the fact that hydrocortisone was permanently discontinued two days before adrenalectomy and she received only enough hormonal therapy to carry her over the operation and immediate post-operative period. Finally, 9-10 days after operation, she started menstruating spontaneously. Bleeding ceased after 3-4 days and she was discharged from the hospital in very good condition.

She has been followed up in both our Gynecology and Endocrine Clinics for over 1½ years. She has been menstruating regularly, at first with the help of cyclic hormonal therapy and, lately, spontaneously. Her vagina has enlarged to normal size although her uterus has remained infantile. Her breasts have developed and all that remains of her former masculine appearance is her low-pitched voice.

She was hospitalized three months ago for re-evaluation and further laboratory studies. Serum Na, K, chlorides, NPN, kepler, insulin tolerance and eosinophile response were all found to give normal results. The 17-ketosteroids, however, have remained in the range of 20-26 mg. in 24 hours.

The greatest subjective improvement has occurred in this patient's personality which, although it had always been very feminine and with normal heterosexual inclinations, was gainsaid by the masculine appearance. After treatment and especially since operation, she has gradually outgrown her initial handicap and now enjoys the normal life of a girl her age. At the last clinic visit she announced her forthcoming marriage.

DISCUSSION

The pathologic lesion associated with the adrenogenital syndrome is a diffuse hyperplasia of the adrenal cortex in no way distinguishable from that associated with certain cases of Cushing's syndrome. Contrary to the latter in which there is an excess of all the known adrenocortical factors, patients suffering from the adreno-genital sydrome show clinical and laboratory evidence of increase in the androgenic factors only. As a matter of fact, in some cases there is even a suggestion of deficiency of the electrolyte and/or the carbohydrate regulating factors. To explain this apparent discrepancy Bartter and his co-workers[3] and Hechter et al[4] have postulated the following theory:

1. There is a congenital abnormality of the adrenal cortex which makes it incapable of elaborating sufficient hydrocortisone due to a block in the transformation of 17-hydroxyprogesterone to hydrocortisone (Fig. 2). This has the dual effect of producing an abnormally large amount of androgenic metabolites of 17-hydroxyprogesterone and creating a deficiency of hydrocortisone.

FIGURE 2

Reproduced from Bongiovanni, A., et al, Adrenal Steroids in Adrenogenital Syndrome, Jour. of Clin. Endocrinology and Metabolism, April, 1954.

2. As a consequence of the latter, the anterior pituitary responds with an increased secretion of ACTH.

3. Increased ACTH then induces cortical hyperplasia with the additional production of androgens.

4. The excessive production of androgens finally causes virilization.

Let us now examine the evidence for and against this theory. In line with such reasoning are the following findings:

1. Sydnor et al[5] have shown high blood levels of ACTH in this disease.

2. Kelley[6] and Goldberg[7] found consistently low levels of 17-hydroxycorticosteroids in blood and urine in several cases. Bongiovanni[8] reports the same findings in seven of nine cases plus the fact that in seven out of nine the low level of corticosteroids failed to increase under the administration of exogenous ACTH (In the other two cases it was possible to increase the level of corticoids and this has been interpreted by Bongiovanni as suggestive of a partial block in "biosynthesis" which may be overcome with adequate stimulation).

3. The findings by Wilkins,[2] later confirmed by many others[6,7,8] that cortisone administration caused a marked reduction in the excretion of 17-ketosteroids along with clinical improvement and a return to the original condition upon discontinuation of therapy, are additional evidence in favor of the Bartter hypothesis.

4. Finally, Bongiovanni[8] has also shown that pregnanetriol, a probable metabolite of 17-hydroxyprogesterone, is regularly present in the urine of untreated patients and is suppressed by adequate cortisone therapy. It disappears following the administration of ACTH or 17-hydroxyprogesterone, something that does not happen in normal individuals.

On the other hand, there are some observations which are not in accord with the theory of defective biosynthesis. These are the following:

1. The majority of the patients with the adrenogenital syndrome do not show evidence of deficiency of glucocorticoids. Many patients do have normal blood and urinary levels of these corticoids.

2. It seems improbable that a large portion of the 17-ketosteroids, which are of the 11-oxygenated variety, represent metabolic products of 17-hydroxyprogesterone. Jailer (as quoted by Bongiovanni[8] has demonstrated that the 17-ketosteroids obtained after administration of 17-hydroxyprogesterone are not of the 11-oxygenated type.

3. The improvement of patients subjected to partial or subtotal adrenalectomy would be another piece of evidence which would not fit into the picture since this would only accentuate the glucocorticoid deficiency without correcting the fundamental metabolic error.

As far as treatment is concerned, there is no question about the beneficial results of cortisone. However, once we accept cortisone as substitution therapy we are faced with the problem of continuous, indefinite administration. This is not only theoretically

sound reasoning but it has also been demonstrated in many cases including our own that discontinuation of the drug causes a relapse. The complications incident to prolonged administration of cortisone are well known and need not be repeated here.

Surgery, on the other hand, has no rational basis as explained above, yet, when confronted with the alternative of continuous suppressive therapy with rather large doses of cortisone, we wonder if the patient is not better off with one-fourth or less of adrenal tissue and considerably smaller doses of substitutes if symptoms of electrolyte or carbohydrate disturbances should develop.

The answer to the question of what will eventually be the treatment of choice in this disease must await further observations and experimentation. In the meantime, we must be satisfied with the slow but definite progress in that direction.

SUMMARY

A case of female pseudohermaphroditism of adrenocortical origin is presented. Results of the suppressive treatment with hydrocortisone and apparently curative effects of subtotal adrenalectomy are reported. The theories of physiopathology are briefly discussed and evidence is presented both for and against a congenital defect in biosynthesis of hydrocortisone. The therapeutic possibilities are discussed in the light of theoretical and practical implications.

Acknowledgements:

The author gratefully acknowledges the kindness of the following persons and institutions: Merck and Co., Inc., Rahway, New Jersey, for generous supplies of hydrocortisone which made this study possible; F. Landrón-Becerra who performed all the 17-ketosteroids, sodium and potassium determinations; Dr. A. Mejía-Casals who performed the presacral air insufflation, and Dr. R. Marcial-Rojas who prepared frozen sections of the adrenals at operation and performed the complete pathologic study.

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RADIOACTIVE ISOTOPES

*CARLOS F. JIMÉNEZ TORRES, M.D.**

Interest in radioactivity dates back to 1896, in which year A. H. Becquerel announced before the Paris Academy of Sciences the results of his discovery of radioactive radiations emitted by uranium compounds. Physicists became very interested in these radiations and soon it was discovered that they consisted mainly of alpha-rays (or helium nuclei), beta-rays (actually electrons) and gamma-rays, of electromagnetic nature and in many respects similar to light waves.

All elements with atomic weights greater than 209, or atomic numbers greater than 83 are naturally radioactive; in other words they possess the property of spontaneous disintegration. The best known example of this property is the formation of radium and radon from uranium.

Later it was possible to produce radioactive forms of all the elements of the atomic table by artificial means. Some of these have been found useful in medical diagnosis and therapy. In the last two decades, "nuclear medicine" has been the object of thousands of research projects with the result that already radioisotopes have certain definite applications in modern diagnostic and therapeutic methods.

Most, if not all of you, have come in contact with the increasing reports in the medical literature concerning the so-called tracer studies, by which the radiations from radioisotopes administered to human beings are measured in various ways. By this method great advances have occurred in the understanding of circulation, the metabolism of body fluids and electrolytes and other important physiologic processes. It is well known that estimates of thyroid function by the use of radioiodine (I 131) are the most accurate so far available. We shall chiefly concern ourselves about the therapeutic applications of these substances.

Of the isotopes used in therapy, a small group merits special consideration:

RADIOPHOSPHORUS

This isotope has been found most useful in the treatment of polycythemia rubra vera and the chronic leukemias. It is administered in the form of sodium acid phosphate, either intravenously or by mouth. P 32 emits B radiation and has a half-life of 14.3 days.

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* Presented before the Southern District Medical Society, August, 1954.

** From Clinical Dr. Pila, Ponce, P. R.

The chief complication which may occur from its administration is severe depression of the hematopoietic tissue in cases of overdosage or of unusual sensitivity to the substance. Leukopenia is most common, but fortunately seldom serious. Thrombocytopenia or anemia may at times be severe enough to require transfusions.

In true polycythemia a definite symptomatic relief invariably follows administration of P 32. This response may occur from a week to a month after treatment. A reduction in the hematocrit is usually apparent in 2 weeks, with a normal hematocrit usually within 3 months time. The remissions may last from 6 months to two or three years, averaging 18 months.

The indications for the use of this isotope in the treatment of chronic myelogenous or lymphatic leukemia are the same as those for roentgen therapy. These are, in general: (1) systemic symptoms such as fever and malaise; (2) enlarged spleen, and lymph nodes giving pressure symptoms; (3) increased leukocyte count associated with increasing anemia or thrombocytopenia; (4) symptoms of increased metabolic rate. The isotope, in the leukemias, may be used as a substitute for, or as a complement of, roentgen therapy. P 32 may be preferred over X-Ray therapy because of its ease of administration and dose control and by the usual absence or radiation sickness and other side effects.

Radiophosphorus has been used as adjunct therapy in the management of selected cases of malignant lymphomata. The results of its use in multiple myeloma are yet uncertain, even if dramatic results have occurred occasionally. A short series of casas of breast metastases to bone has been treated with enocuraging results.

RADIOIODINE

I 131 is a fission product of uranium and may be obtained in a carrier — free form from the A. E. C. at Oak Ridge. It has a half-life of 8.0 days and emits B and gamma radiation. The substance, administered in the form of sodium iodide has found its greatest use in hyperthyroidism. Certain advantages over surgery are the absence of mortality and other complications of surgical intervention, such as damage to the parathyroids or the recurrent laryngeal nerves. It has been reported that by this method of management malignant exophthalmus and recurrences of hyperthyroidism are less frequent.

As in surgery, myxedema is the important complication of treatment. It can be usually avoided by fractionation of the dose. The results of the use of radioiodine in thyroid carcinoma are variable, depending on the histopathology of the tumor and the

rate of uptake, or the avidity of the cells for the iodine. Better results are obtained in struma and follicular carcinoma than in papillary or alveolar carcinoma.

Metastatic lesions have exhibited a greater iodine uptake after removal of the thyroid gland. The administration of pituitary thyrotropic hormone has also raised the iodine uptake of metastatic lesons.

RADIOCOBALT

Cobalt 60, produced in the uranium pile by neutron bombardment of Co 59, emits a weak beta particle and gamma photons of greater energy than the average of radium. It has a half-life of 5.3 years. It is a relatively inexpensive source of radiation, and already there are several teletherapy units which may substitute high-powered roentgen therapy equipment. The isotope can be placed in needles and tubes similar to those of radium. The interstitial method of suturing cobalt containing nylon threads to tumors and tumor beds is increasing in importance. Some observers go as far as believing that cobalt 60 may substitute radium in our therapeutic armamentarium.

RADIOGOLD — (Half Life - 2.7 days)
(B and gamma emitter)

Gold 198 has been often used (in colloid suspension) inside serous cavities in order to control metastatic implants and to prevent further formation of malignant effusions.

Satisfactory results have been obtained by the injection of a solution or radiogold in inoperable tumors such as the prostate. The colloid has a tendency to remain within those tissues where it is injected.

The use of radiogold in carcinoma of the cervix with pelvic spread is still in a very experimental stage.

The isotope has been used intravenously with fair success in cases of lymphosarcoma and leukemia.

In a metallic form it has lately been used in the same form as radon seeds. The isotope is placed in thin tubes of stable gold. The tubes may be inserted in a planned fashion inside various tumors.

A word of warning in regards to the uses of these substances as in the case of roentgen ray equipment, radium and other sources of ionizing radiations. We must always be aware of the inherent dangers of overexposure, both to our patients and to the personnel handling them.

Isotopes can only be used by those physicians who are adequately trained in radiation therapy and by assistants specially prepared in the technicalities of this mode of therapy.

To conclude we have only attempted to point out the high-lights of the medical uses of some radioactive isotopes.

These new developments of the so-called atomic age, in our opinion, have not quite yet revolutionized our currents concepts of the management of malignant disease. Isotopes, however, have already obtained a definite status of importance in modern medicine. We are certain that their significance will progressively increase with the constant new developments of dynamic medical research.

CLINOCOPATHOLOGICAL CONFERECENCE

PRESBYTERIAN HOSPITAL

*JOSE A. DE JESUS, M.D.**
*RAUL MARCIAL ROJAS, M.D.***

Clinical Summary: A sixty-five-year-old white woman was admitted to the Presbyterian Hospital on March 24, 1955. She was a well known diabetic complaining of constipation and inflammation around the anal area of three months duration. Her lifelong constipation had increased lately. The constipation was accompanied by pain and irritation around the anus on defecation. Her family history was non-contributory. A cyst was removed from the vagina in 1938, the etiology of which was unknown. She was submitted to a cholecystectomy in 1953. There were no other symptoms referable to any of the other systems except for the genito-urinary one. She complained of frequency and slight dysuria. She had a urinary infection before admission for which she received sulfanilamide. The exact dosage was not known. Her last menstrual period was ten years before admission and since then no vaginal bleeding had occurred.

Physical examination revealed a well developed, well nourished elderly woman in no acute distress. The blood pressure was 150 systolic, 70 diastolic. The pulse was 90, respirations 25 and temperature 99.6°F. The only postive finding encountered was that of external hemorrhoids. The pelvic examination was done on March 25, 1955 by a gynecological consultant who did not encounter any abnormalities of the genital system. He felt an indurated mass in the region of the urethra and suggested an urological consultation.

The patient was having an average of three diarrheic bowel movements consisting of very liquid stools for the first three days after admission. She was quite restless and nervous and, according to one observer, showing definite senile mental changes. The general condtion of the patient gradually deteriorated as well as her mental status.

From admission till six days before death she was running a low grade fever. Urine culture was positive for B. Coli in March 29, 1955. The latter proved to be sensitive to chloromycetin. She was given this drug. On April 2, 1955 the urinary output was

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* By invitation. From Department of Medicine, San Juan City Hospital and School of Medicine, School of Tropical Medicine, University of Puerto Rico.
** From Department of Pathology, Presbyterian Hospital and School of Medicine, School of Tropical Medicine, University of Puerto Rico.

markedly decreased and bloody. Non protein nitrogen was 154 mg. %. A urological consultation on April 2, 1955 entertained the diagnosis of lower nephron nephrosis and treatment was recommended for this condition. The patient was started on 10% glucose in water intravenously. Later at night on April 2nd she was considered to be almost anuric with diminished chloride and sodium values with slight increase in potassium. Lactate solution 20% was given by mouth. She vomited once or twice but apparently her state of hydration was adequately maintained. The patient rapidly deteriorated, became markedly drowsy and went into complete renal shut down on April 6, 1955. Gastrointestinal lavage was established. At 9:00 A. M. the patient was cold, clammy, and pale. The blood pressure was 90/70. Blood, oxygen and coramine were immediately ordered but the patient expired quietly at 11:00 A. M.

Laboratory Data: Blood: 3/24/55 - RBC 3,520,000; Hb. 76% (11 grms); C. I. 1.0; WBC 16,350; Diff. PMN 84; Stabs 8; Lymphos. 6; Monos. 2. **Urinalysis:** 3/24/55: SG 1.001; Alb. and Sugar: Negative. WBC 3-5 p.h.p.f. RBC O-2. 3/27/55: Many clumps of WBC. Urine culture: 3/26/55 - Positive for B. coli. Sensitive to chloromycetin. **Feces:** 3/25/55 - Ova of Trichuris and Ascaris. **Blood chemistry:** 3/28/55 Glucose 115 mgr. %. 4/1/55: Glucose 85% mgr. %. Chlorides 500 mgr. % (85 mEq/1.) NPN 154 mgr. %. 4/2/55: Sodium in serum 127 mEq/1. Potassium in serum 8.4. 4/4/55: Glucose 105 mgr. % Chlorides 500 mgr. % (85 mEq/1.) NPN 32 mgr. %. Potassium in serum 8 mEq/1. 4/5/55: NPN 160 mgr. %.

Discussion:

Dr. De Jesús: At age 65 degenerative disorders and neoplasia must be considered in every case. The patient being a diabetic will bring to the foreground arteriosclerosis with its complications. She certainly has a blood pressure typical of arteriosclerosis of the large vessels, particularly of the aorta. Evidence will have to be weighed in pro and con of Kimmelstiehl-Wilson disease, necrotizing papillitis, acute and chronic pyelonephritis and cystitis. Any of these conditions are to be expected in a diabetic of 65 years of age.

Chronic constipation has no significance per-se but when it has become progressive one must consider an organic lesion particularly a neoplastic one of the large bowel. In this lady however the anal irritation might have been responsible for this as the act of defecation made the anal irritation worse. This inflammation around the anus could very well be due to a Monilial infection, common in diabetics or to the hemorrhoids which are frequently ac-

companied by itching when fissures are present. When thrombosed hemorrhoids are very painful.

The frequency and dysuria certainly point to bladder or urethral irritation most likely from infection. This lady was actually treated for urinary infection prior to her admission and then again during her hospital stay. Urinary infections are common in diabetics and I doubt seriously that those were the only instances of urinary infection in this lady. She must likely had them repeatedly over a number of years.

There was an important event in this patient's illness, she received sulfanilamide. This may bring about very serious conditions such as polyarteritis and other sensitivity states and renal damage, lower nephron nephrosis being a prominent one. The latter is usually preceded by microscopic hematuria and heavy crystalluria. This patient receivevd chloromycetin shortly after admission. According to the nurses' notes the urinary output was 350cc on March 30; 125cc on April 1st and 54cc on April 2. Thus the patient was actually admitted with a low urinary output which was gradually progressive denoting a gradual renal shut down rather than a rapid one.

Therefore I can not blame Chloromycetin for the renal shot down as the disminution of urinary output was already under way when the administration was started. How about the sulfonamide? A very good possibility. Unfortunately I do not know when she took her last dose.

Dr. Paniagua: Her last dose was ten days prior to admission.

Dr. De Jesús: That is very helpful information indeed. This drug could have caused a lower nephron nephrosis but there are certain facts that tend to rule this out. The shut down progressed slowly and occurred rather late in relation to time of dosage. There were no crystals in the urine, and the urine became bloody late, rather than early. The specific gravity was 1.001.

In lower nephron nephrosis a diminution in urinary output is noted within 24 hours of the renal insult. The urine as it is passed, is usually acid and the specific gravity 1.010. In many instances the urine may be frankly bloody or smoky for the first day or two. Proteinuria of varying degree occurs early and persists throughout illness. The blood pressure goes down initially, then becomes normal and thereafter goes up to definite hypertensive levels. Could this be due to Kimmelstiehl-Wilson disease on its terminal stage? Yes, it could be but there is too much evidence against it. There was no proteinuria which is moderate to massive in this condition. No laboratory evidence nor clinical evidence of hypoproteinemia is offered in the protocol. Edema and hy-

pertension were absent. Glomerular nephritis is ruled out on the same basis as Kimmelstiehl-Wilson disease as clinically they behave very similar. Diminished sodium, diminished chlorides and increased potassium is seen most often in lower nephron nephrosis particularly during the early stages and occasionally in uremia from other causes. Then, what else could give us such a picture? Polycystic kidneys could be responsible for such a state of affairs. but no observer could palpate the masses. Besides there are too many other features that can not be explained on the basis of this diagnosis. It could have been an obstructive uropathy. The gynecologist has ruled out Ca. of the cervix with ureteral obstruction for us. Up to this point I can not establish with certainty which one of the conditions I mentioned was responsible for the renal shut down.

Even though the protocol records bloody urine on April 2, 1955, the nurses recorded bloody urine from March 31st on. Bloody urine to me means gross hematuria. The golden opportunity to find the source of hematuria is to examine the patient while the bleeding is going on. If one postpones the examination until the next day, the bleeding may cease and no obvious cause for it may be found. In such instances, all one can do is prove or attempt to prove the normality of the urinary tract or at least exclude the presence of malignant disease. This is not as gratifying as to be able to observe a spurt of bloody urine issuing from one or the other ureteral orifice. Thus at this point I believe a cystoscopic examination would have been of paramount importance and would have probably given us the diagnosis. Unfortunately this was not done and one is forced to go on the evidence afforded by the protocol. The differential diagnosis of hematuria entails a prolonged discussion. Suffice it to say that it may be caused by infection, stones and tumors anywhere along the genito-urinary tract as well as by systemic conditions which cause bleeding through defects in the clothing mechanism or in the blood vessel structures. Toxins, poisons as well as glomerulonephritis may cause gross hematuria. Therefore it behooves one to find or to rule out evidence in favor of these latter conditions.

The indurated mass in the urethra poses a very interesting situation in this patient. Dilatations of a periurethral duct forming a cyst, and an urethral diverticulum although forming a mass are not indurated unless a stone has formed or lodged in them. Urethral caruncle is usually painful and easily seen. It is soft unless malignant degeneration has taken place (melanoma or carcinoma). A tumor may present itself as an indurated mass in the

female urethra and not uncommonly the tumor may be a pedunculated one coming from the bladder proper.

At 7:30 A. M. the nurses on day of death record no change in the patient's condition. Then at 9:00 A. M. some catastrophe took place. She became cold, clammy and went into shock with blood pressure hovering among 90/70. The physician instituted heroic measures. What could have cause this? Four conditions came to my mind:

1—Cerebrovascular accident.

2—Dissecting Aneurysm with rupture.

3—Massive Coronary Occlusion.

4—Pulmonary embolism.

I shall leave these conditions hanging on fire and tried to reconstruct what I think happened to this patient. The patient had been a diabetic apparently mild judging by the protocol. We know that diabetics are very prond to develop repeated urinary infections and there is evidence for at least one such episode in the protocol. She had evidence of bladder irritability. So she certainly had cystitis. She presented hematuria and anuria together with the tumor in the urethra. On the basis of infections, anuria, gross hematuria and a palpable mass in the urethra one is justify in suspecting tumor of the urinary bladder. The tumors of the urinary bladder are often multiple and it is not unusual for a pedunculated one to prolapse into the urethra. This patient was catheterized without difficulty thus making the possibility of an intrinsic urethra tumor less likely. This bladder tumor obviously invaded the ureteral orifices causing an obstructive uropathy explaining the renal shut down. A bladder tumor likewise explains the rectal pain as it is well known that such tumors may cause pain referrable to the rectum and the vagina. They too may cause diarrhea, particularly in children.

But before she died she suffered a catastrophe manifested by shock. This could have been a massive coronary occlusion which is a very likely possibility, perhaps the best and there is no evidence against it. In a cerebrovascular accident some evidence of paralysis would have become evident. In dissecting aneurysm one likes to have a history of pain of tearing quality migrating down the chest without shock sometime in the past. Then we have pulmonary embolism. Could this have happened? Yes. I think it is a toss-up between this condition and the massive coronary occlusion. Moreover, she was a debilitated patient lying in bed.

A condition which predisposes to thrombophlebitis of the legs and better still this lady had to my mind enough pelvic pathology

to warrant the presence of thrombophlebitis of the pelvic veins. This could very easily account for embolization of the lungs.

Therefore my final diagnosis is:

1—Diabetes Mellitus
  Old pyelonephritis changes
2—Cystitis and a precancerous stage such as
  Leukoplakia,
  Cystitis glandularis or
  Cystitis cystica
3—Ca. of urinary bladder
  Ureteral obstruction
  Hydro or pyoureter
  Hydro or pyonephrosis
  Pedunculated tumor (went into urethra)
4—Pelvic vein thrombophlebitis
  Embolization to lungs
5—Terminal bronchopneumonia — an almost constant event occurring terminally
6—Arteriosclerosis.

Dr. Montilla: Why don't you favor coronary thrombosis?

Dr. De Jesús: I think that is a very good possibility. In fact I already said it was a toss-up between pulmonary embolism and massive coronary acclusion. I favor the former because I think there was enough pathology in this lady's pelvis to make pelvic thrombophlebitis a very good possibility.

Dr. R. A. Marcial-Rojas:

It is a great pleasure indeed, for me as a pathologist, when an able clinician in such a marvelous and well organized presentation almost enumerates all the anatomical diagnoses of the case and their correct sequence. I say it is a great pleasure, because not only he demonstrated his clinical knowledge and acuity but also definitely proved that the case was an ideal one for this type of presentations. To provide cases with such fine correlation between pathologic and clinical findings should be the goal of the pathologist.

This 65-year old diabetic woman presented a severe cystitis glandularis. The latter gave origin to an adenocarcinoma of the urinary bladder, mostly intramural and completely obstructing both ureteral orifices. Bilateral hydroureter, hydronephrosis and pyelonephritis followed. The tumor extended to the vesico-vaginal septum and showed pelvic lymph node matastases. The left hypogastric vein was partially obstructed by a thrombus and exhibited definite evidence of thrombophlebitis.

An embolus migrated to the right pulmonary artery accounting for the final sudden episode and death.

Anatomical Diagnosis:

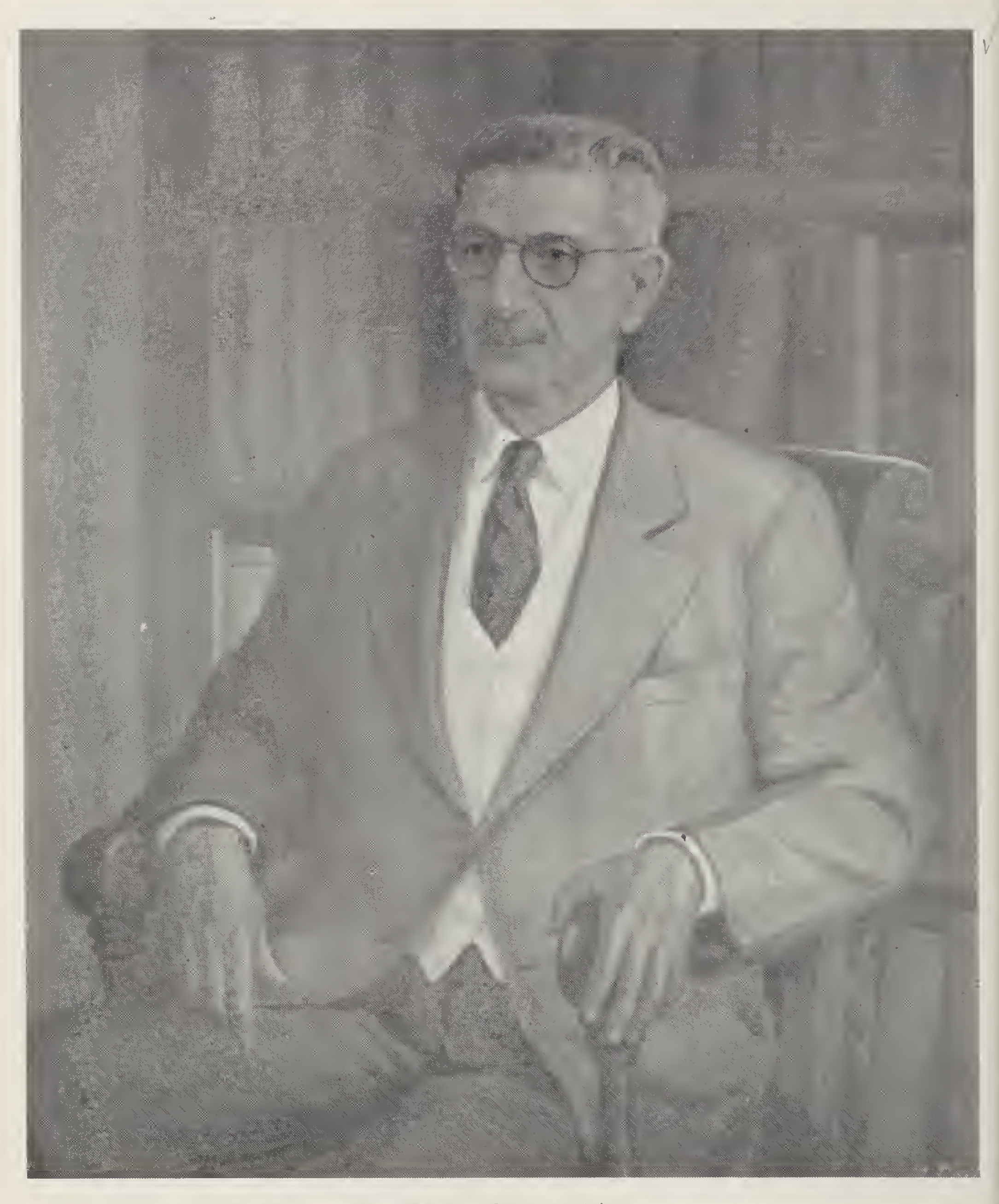
(1) Chronic cystitis glandularis
(2) Adenocarcinoma of urinary bladder with extension to vesico-vaginal septum and metastases to pelvic lymph nodes
(3) Hydroureter and hydronephrosis, bilateral, due to tumoral obstruction
(4) Chronic pyelonephritis, bilateral
(5) Thrombophlebitis of left hypogastric vein
(6) Pulmonary embolism, right pulmonary artery
(7) Focal atelectasis
(8) Atheromatosis of aorta, far advanced
(9) Generalized arteriosclerosis, moderate
(10) Chronic cervicitis

DR. MANUEL QUEVEDO BAEZ
Ha fallecido

Lista ya para entrar en prensa la presente edición del Boletín, en la cual incluímos los discursos pronunciados durante el homenaje que rindiera la Asociación Médica de Puerto Rico, el sábado 22 de octubre ppdo., a su ilustre fundador y primer presidente con motivo de la develación del retrato al óleo que le fuera hecho en fecha reciente, recibimos la triste noticia del sensible fallecimiento de este ilustre médico, acaecido en su residencia en Santurce en la tarde del martes 22 de noviembre.

Sabíamos que don Manolo, como cariñosamente le llamábamos, estaba herido de muerte, desde el momento preciso en que, por primera vez en su larga y fecunda vida, nos presentó sus excusas por no poder trasladarse al domicilio de la Asociación Médica para recibir el homenaje de sus colegas en el acto celebrado el 22 del pasado mes; pero nunca pensamos que nos veríamos privados de hacerle entrega de una de las primeras copias de esta edición para darle la satisfacción de tener un recuerdo más de su querida Asociación.

En nuestra próxima edición publicaremos una nota necrológica del ilustre médico fenecido y dejamos en las páginas que siguen —tal como lo habíamos planeado— el testimonio del cariño y la admiración que siempre tuvieron para el gran amigo desaparecido todos los miembros de la clase médica puertorriqueña.



Dr. Manuel Quevedo Báez

Retrato al óleo que fué develado el día 22 de octubre de 1955 en el domicilio de la Asociación Médica como tributo de admiración y cariño hacia nuestro fundador y primer presidente.

HOMENAJE AL DR. MANUEL QUEVEDO BAEZ CON MOTIVO DE LA DEVELACION DE SU RETRATO EN LA ASOCIACION MEDICA DE PUERTO RICO

PALABRAS DEL PRESIDENTE

Compañeros, Damas y Caballeros:

Estamos reunidos aquí esta tarde para rendirle un tributo más de nuestro cariño y admiración a uno de los miembros más venerados y queridos de nuestra Asociación.

En varias ocasiones anteriores le hemos honrado en distintas formas, pero la actual dirección de la Asociación ha creído que en esta nuestra casa debía existir un recuerdo imperecedero de este compañero que no se conformó con escribir la historia de la medicina en Puerto Rico, sino que hizo gran parte de esa historia al fundar y ayudar a sostener por más de medio siglo esta gran Asociación Médica que es hoy la mayor satisfacción de su vida.

El Dr. Manuel Quevedo Báez es para la clase médica de Puerto Rico un apóstol. Por la sencillez, y la humildad que le han caracterizado durante su larga vida, que Dios guarde, ha sido admirado y querido por todos los que han tenido la dicha de conocerle y de tratarle.

Médico de profundos conocimientos, su consagración a la profesión y su ética intachable son de todos conocidas, así como sus dotes de escritor y literato.

El Dr. Quevedo Báez ha visto tres generaciones de médicos desfilar ante sus ojos de padre de esta Asociación y yo les puedo asegurar que hoy se siente muy contento y orgulloso al ver la pléyade de jóvenes galenos que se suman diariamente a las filas de esta Asociación que él fundara en unión del Dr. Stahl y otros compañeros insignes y de la cual fué el primer presidente.

Yo me sitúo en la segunda de estas tres generaciones de médicos que ha conocido bastante bien al Dr. Quevedo Báez y me siento muy complacido de que nuestra Asociación le brinde hoy este sencillo tributo de amor y veneración.

Muchas gracias.

Ricardo F. Fernández, M.D.

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EN LA DEVELACION DEL RETRATO
DEL DR. MANUEL QUEVEDO BAEZ

El Dr. Guzmán Rodríguez dijo . . . .

Asistimos a un acto de justo reconocimiento, a la figura prócer del fundador y primer presidente de la Asociación Médica de Puerto Rico. El Dr. Manuel Quevedo Báez, quien a fuerza de ta-

lento, gentileza y austeridad se volvió noble, representa en estos momentos, "la dichosa edad y siglo dichoso aquel" de románticos hidalgos, "de lanza y astillero y adarga antigua" que creyeron que el placer del deber cumplido, bien podía comprarse al precio de la escasez y de la miseria.

Por esas razones, en esta otra edad y en este otro venturoso siglo, cuando las lanzas y las adargas sólo se vislumbran en las salas abandonadas y solitarias de los museos, vemos sin embargo la figura prócer del último de nuestros hidalgos, encorvado por los años pero erguido por la austeridad, que en su andar trémulo y vacilante, vive desgranando el rosario de la pobreza y musitando la oración de la dignidad.

Hemos dicho en múltiples ocasiones que la historia de Puerto Rico no podría escribirse si se soslaya la influencia que la pluma de sus médicos ejerciera a despecho de las intransigencias coloniales, en el desarrollo de las ideas y en la defensa y mantenimiento de los ideales.

Betances y Alonso, Padilla y Zeno Gandía, Barbosa y Gómez Brioso, Guzmán Rodríguez y Martínez Roselló, Font y Guillot y Quevedo Báez fueron entre otros muchos los portavoces militantes del vía crucis dolorosa del criollo.

En la rutina de aquella devoción en que la elevación del carácter y práctica de las virtudes ciudadanas, eran timbres reconocidos de nobleza, los médicos oficiaban en el templo del periodismo y fueron sus tribunas: Borinquen de Betances, La Correspondencia de Zeno Gandía, El Tiempo de Barbosa, El Laboratorio de Guzmán Rodríguez, La Verdad Médico Social de Font y Guillot y El Palenque de Quevedo Báez.

De ese grupo de irreductibles paladines, sólo nos queda,

...Don Manuel Quevedo Báez...

Hemos pronunciado su nombre envolviéndolo en la penumbra del susurro, para que nadie, absolutamente nadie, al enterarse de que aún vive, nos lo quiera arrebatar. Bien puede decir Quevedo Báez remedando al ciego de Esmirna y recordando aquella generación de desaparecidos titanes:

"Desciendo de esa sangre y me glorío."

Desde el año 1894, Quevedo Báez inicia su obra médico social en Puerto Rico, campeando a veces como uno de los más atildados prosistas de la literatura regional y otras veces clamando desde la tribuna del Ateneo, del cual fué eximio Presidente, por un Puerto Rico mejor para las clases desheredadas de la fortuna.

Hombre incapaz de avenirse al yugo de hipócritas claudicaciones en complicidad con los vanos halagos, llegó a los altos puestos por méritos propios, ya que deslindando con austeridad los cam-

pos de la politiquería, había sido señalado como un inveterado izquierdista de peligrosos matices radicales.

Así fué Secretario del Tribunal de Médicos Examinadores desde su fundación y Presidente hasta el 1931; fundador y Presidente de la Asociación Médica de Puerto Rico hasta el 1904; médico de la Policía Insular durante un cuarto de siglo hasta el 1950 y Director de Higiene Escolar de San Juan hasta el 1927.

Fué un devoto servidor público de recio temple, habiendo desempeñado todos los puestos desde los humildes de médico titular de Puerta de Tierra y visitante del Lazareto, hasta la presidencia de la Asociación Médica de Puerto Rico. A todos ellos llevó su elegante austeridad y los puestos más humildes dejaron de serlos, cuando fueron por él desempeñados.

Gran parte de la obra científica y literaria de Quevedo Báez vió la luz pública en El Boletín de la Asociación Médica, Heraldo Español, correspondencia de Puerto Rico, Puerto Rico Ilustrado y El Palenque de la Juventud.

Indice de la más tesonera voluntad en la literatura y en la ciencia médica regional, lo fué sin duda nuestro ilustre amigo, quien en su obra polifacética enfocó todos los problemas de su tierra con amor y valentía.

Sería vano alarde o injustificado despropósito, intentar un estudio bibliográfico, no empece lo minúsculo, de la obra científico literaria de Manuel Quevedo Báez.

Pero alarde o despropósito, no sería posible ignorar ni menos silenciar el aporte que relevante y generoso hiciera nuestro dilecto amigo, a la causa de la liberación del isleño, consumido por la miseria y roído por la ignorancia.

Los estudios que sobre medicina forense, terapéutica social del delito, métodos para combatir el desarrollo del alcoholismo, sicología infantil y relaciones sociales entre el hogar y la escuela, fueron trabajos documentados, honra y prez de la sociología y de la sonora lengua castellana.

Pero el Dr. Quevedo Báez fué además de literato insigne y maestro indiscutible del buen decir, higienista notable de relevante cultura científica, que supo enfocar al mismo tiempo los problemas sanitarios de su tierra, en sabias disertaciones sobre la fiebre amarilla, tuberculosis infantil, prevención de la uncinaria, protección a la infancia en la lucha antituberculosa y gastro enteritis infantil.

Vamos a develar el retrato del Dr. Quevedo Báez. Vamos una vez más a homenajear al médico insigne que habiendo legitimizado su derecho a la gloria y al recuerdo, a través de medio siglo de luchas sin cuartel, escribe en sus últimos años, cuando estaba jus-

tificada la levedad del espíritu y el legítimo descanso, la historia de la medicina en Puerto Rico.

La Asociación Médica de Puerto Rico está en deuda con el Dr. Quevedo Báez. Los esfuerzos titánicos de los últimos años para solventarla, han sido dignos de encomio, pero la magnitud de la deuda ha sobrepasado los esfuerzos de los deudores.

El Colegio de Abogados de Puerto Rico en un acto de comprensiva solidaridad, estableció para Don Roberto H. Todd, una secretaría vitalicia.

Y nosotros, ante la magnitud de la deuda, ¿qué vamos a hacer nosotros, por nuestro Dr. Manuel Quevedo Báez?

Que no se diga como en ocasión memorable dijera Rufino Blanco Fombona, que

> "En América debemos convencernos que no es bastante producir varones ilustres, que es necesario merecerlos."

* * *

PALABRAS DEL DR. O. GARCIA QUEVEDO

Es para mi un verdadero privilegio y un honor inmerecido, encontrarme en la tarde de hoy participando en acto de tan significada magnitud y trascendencia. Pero gracias al deseo expresado por el homenajeado, para mí, mandato ineludible, exteriorizado en los estrechos y limitados espacios de un telegrama, y luego reafirmado personalmente, cumplo a la altura de mis escasos y pobres facultades ese deber: complacer al Dr. Manuel Quevedo Báez, mi dilecto tío, padrino y padre espiritual, representándolo en este acto solemnísimo.

Dicen los sociólogos organicistas que los hombres, al igual que los pueblos y sus Instituciones, son organismos simples, con parecidas modalidades funcionales, taras, degeneraciones, etc. Y que son eternamente jóvenes, mientras vivan la etapa del yo personal, pero éste tiende a acoplarse y es el carácter el que busca y propicia esa conjunción, que al verificarse, les imprime el sello de la "personalidad" dejando de ser imitativos, desoyen ajenas sugerencias para tornarse "autónomos". En cuanto al hombre, este deja de ser esbozo, y adquiere los relieves maravillosos de la "personalidad", llevando entonces en su mente, la brújula que le orientará en el curso que seguirá en su diario desenvolvimiento. Son ellos los que al caminar dejan sobre el sendero sus huellas bien profundas a su paso por la vida. Deseó siempre el Dr. Quevedo Báez, la Casa del Médico, pero como el deseo es algo vago que nos inclina al objeto que lo provoca —desear es una manifestación subconsciente— fué más allá y se adentró en el querer. Querer es la concentración de la mente en una visión única, traducida luego en la compactación de todo el Ser en una sola ejecución, y como el que

sabe querer, sabe también obrar, he aquí su fruto: Esta Casona que acoge reverente a los apóstoles del dolor, para unirlos en la comprensión, en la amistad y en el compañerismo. Como honrar honra, los que hoy conllevan la responsabilidad de la dirección de clase tan selecta; de esta Asociación Médica de Puerto Rico, ponen de relieve una vez más su condición de madurez como agrupación, exaltando la personalidad de su fundador y primer presidente el Dr. Manuel Quevedo Báez, reconociéndole su gesta, colocando en sitio privilegiado la vera efigie de su persona, en ese óleo magistral, que copia con exactitud los perfiles de su figura, y que servirá para decirles a las generaciones presentes y a las venideras, que gracias a su deseo, a su esfuerzo y a su actuación, se levanta para orgullo y honra de Puerto Rico este sitial, que a semejanza de los templos, debe siempre conservar su pristina pureza: que se queden de puerta afuera, los egoísmos, las envidias, los odios y las malquerencias, y que impere supremo el amor en todos sus recintos. Es así como lo concibió Quevedo Báez, y es así como deben mantenerla, los que tienen el alto privilegio de frecuentarla. Manolo (permítaseme así llamarlo) es un apasionado luminoso que borra los defectos al amigo; exalta su desprecio por todo cuanto juzga mezquino; jamás exagera elogios a lo que estima grande; es en fin, de los que reconocen que en el vivir no se cosechan delicias solamente, ya que cada placer tiene su sombra en el dolor, y sabe que el auténtico y legítimo deber del hombre, es el de comportarse en cada instante, del modo que pueda propiciar el mayor bien al mayor número de sus semejantes.

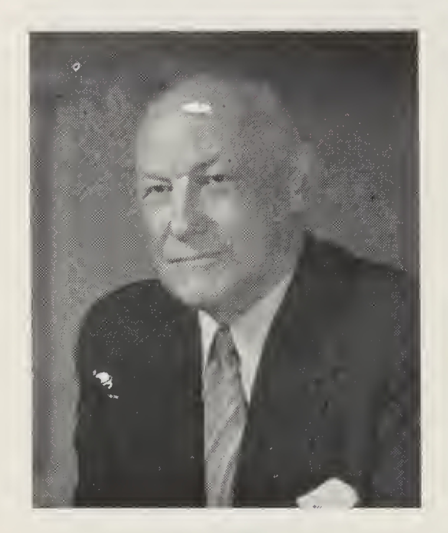
Ojalá y sigais ascendiendo por los escalones de alabastro de la escalera espiritual que os llevarán a ese ansiado ideal de progreso y perfeccionamiento que anida en la mente de todo ciudadano íntegro tan digno de ustedes y de esta maravillosa Asociación Médica de P. R. Creo firmemente en vuestro presente, grande y admirable, pero estoy más seguro de vuestro gran porvenir.

Si logran Uds. como hasta el presente imprimirle a esta Asociación Médica las características de una agrupación, en que la más acabada ética, sea obligada urdimbre de cada socio y la amistad y el compañerismo, características inviolables en su seno, llevarán al alma y al corazón del homenajeado, que por tener un alma grande, acumula gran cantidad de dolores, entre ellos, su ausencia obligada a este acto enaltecedor para su persona, el homenaje espiritual más halagador y el más preciado reconocimiento. Y como él tiene erigido un altar en mi corazón, que lo ocupa por completo, me autoriza decirles a todos en su nombre: Gracias, muchas gracias por este acto de hoy.

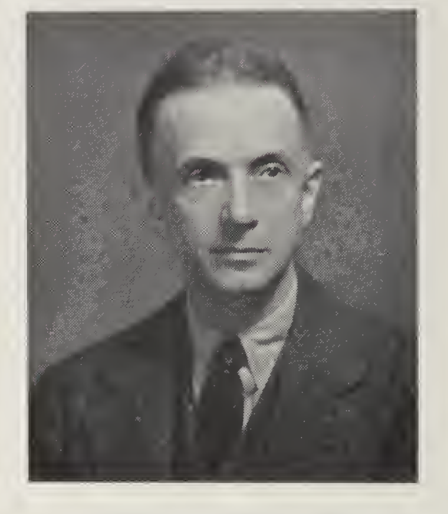
PROGRAMA DE LA QUINCUAGESIMA SEGUNDA ASAMBLEA ANUAL DE LA ASOCIACION MEDICA DE PUERTO RICO

Diciembre 6-10, 1955

HUESPEDES DE HONOR



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Presidente de la Asociación Médica Americana; Jefe de la Facultad, St. Vincent's Hospital; Jefe del Departamento de Urología, Hamot Hospital, Erie, Pa.



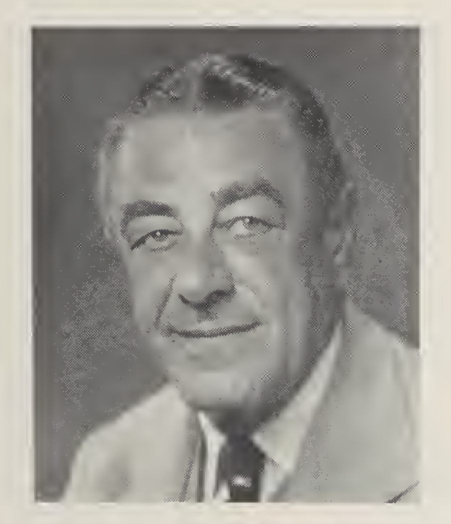
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Profesor y Director del Departamento de Patología, Escuela de Medicina de la Universidad de Johns Hopkins, Baltimore, Md.



BERNARD J. ALPERS, M.D.
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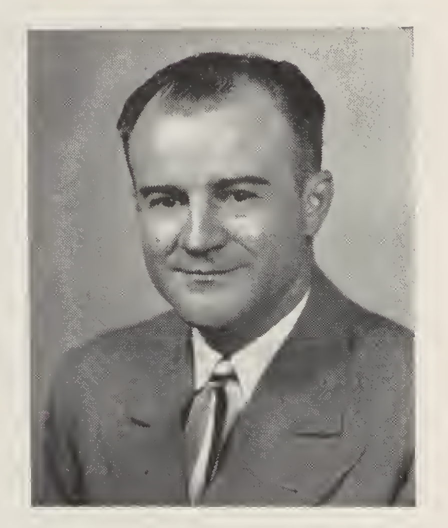


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Profesor de Medicina Interna y Director de Endocrinología y Metabolismo, Escuela de Medicina de la Universidad de Michigan, Ann Arbor, Michigan.



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WILLIAM F. FINN, M.D.
Profesor Asistente Obstetricia y Ginecología, Colegio Médico de Cornell, New York City.

CAMARA DE DELEGADOS

Reuniones Administrativas

Sábado, 3 de diciembre, 2:00 p.m.

La Cámara de Delegados celebrará su primera reunión a las 2:00 de la tarde del sábado, 3 de diciembre, en el salón de actos de la Asociación.

A esta reunión deberán asistir, además de los miembros de la Cámara, los presidentes de los comités permanentes de la Asociación. Los asuntos se discutirán en el orden siguiente:

(a) Llamada al orden
(b) Constitución de la Cámara
(c) Lectura y aprobación de informes
(d) Asuntos a tratar

Martes, 6 de diciembre, 2:00 p.m.

La segunda reunión de la Cámara de Delegados se llevará a efecto en el domicilio de la Asociación el martes, 6 de diciembre, a las 2:00 de la tarde, y se observará el siguiente orden:

(a) Llamada al orden
(b) Informe del Comité de Nominaciones
(c) Elección de funcionarios para el año 1956.
(d) Asuntos a tratar

—

MARTES, 6 DE DICIEMBRE

Hotel Caribe Hilton
8:00 p.m.

Presidirá el Dr. Agustín M. de Andino, Jr.

1. Palabras de Bienvenida, Agustín M. de Andino, Jr., M.D.
2. Entrega de premios
   (a) Medalla "Dr. Manuel Quevedo Báez"
   (b) Medalla al mejor estudiante de medicina de la Escuela de Medicina de la U.P.R.
   (c) Premio al mejor trabajo publicado en el Boletín de la Asociación Médica de Puerto Rico - año 1955.
3. Discurso presidencial, Ricardo F. Fernández, M.D.
4. Palabras del Presidente de la Asociación Médica Americana. Elmer Hess, M.D.
5. Recepción presidencial.

MIERCOLES 7 DE DICIEMBRE

Hospital Presbiteriano
(Casa de Enfermeras)
9:30 a.m.

Presidirá el Dr. Dwight Santiago

9:30 a.m. Dextrocardia and Situs inversus totalis - Report on four cases in one family.
Dwight Santiago-Stevenson, M.D. and
Pedro Ortiz-Santiago, M.D.

9:55 Thrombophlebitis Migrans associated with Neoplasia.
Roberto Jiménez-López, M.D.

10:20 Intermission

10:35 Obstetrical Hemorrhage and Hypofibrinogenemia.
Armando García-Castillo, M.D.

11:00 Pseudomyxoma Peritonii due to Ruptured Appendicial Mucocoele with Carcinoid Tumor - Report of Case.
Pedro A. Suau, M.D. and
Raúl Marcial, M.D.

MIERCOLES 7 DE DICIEMBRE

Escuela de Medicina
(Auditorium, Puerta de Tierra)
2:30 p.m.

Presidirá el Dr. E. Harold Hinman

2:30 p.m. Symposium: Certain Aspects of Anemias as Seen in Puerto Rico.
(a) Nutritional Anemias
A. A. Cintrón-Rivera, M.D., R. S. Díaz-Rivera, M.D., and Miss Josefina Acosta-Matienzo, M.S.
(b) Sickle-cell and related hereditary anemias.
Antonio Ortiz, M.D., M. Vicente de Torregrosa, Ph.D. and A. Rivera Trujillo, M.D.
(c) Anemias of Pegnancy.
M. Fernández-Fuster, M.D., C. R. García, M.D., and A. Cintrón-Rivera, M.D.
(d) The Role of Surgery in Puerto Rican Anemias.
J. Noya-Benítez, M.D. and F. Raffucci, M.D.
(e) Discussion.

4:00 p.m. Clinical Study of Urosulfin.
Luis A. Sanjurjo, M.D.

4:20 p.m. Technique of Dacryocystorhinostomy. (Illustrated by motion picture) Guillermo Picó, M.D.

MIERCOLES 7 DE DICIEMBRE

Asociación Médica
8:30 p.m.

Presidirá el Dr. Arturo L. Carrión

8:30 p.m. Reparación plástica del párpado superior tras excisión de carcinoma (película).
Ricardo F. Fernández, M.D.

8:45 p.m. Clinical Implications of Aldosterone, the Newly Recognized adrenal Steroid. Jerome W. Conn, M.D.

9:30 p.m. The General Practitioner as an Urologist.
Elmer Hess, M.D.

JUEVES 8 DE DICIEMBRE

Hospital de la Capital
(Anfiteatro, Dispensario Escuela de Medicina)
8:30 a.m.

Presidirá el Dr. Fernando A. Battle

8:30 a.m. Ward rounds, all services.

9:00 The Problem of Urethrotrigonitis in Women.
Roberto Fortuño, M.D. and
Benigno Rodríguez-Lucca, M.D.
Discussion: Luis A. Sanjurjo, M.D.

9:20 Sickle cell Anemia complicating Pregnancy.
Paul Mari-Rodríguez, M.D.
Discussion: Manuel Fernández-Fuster, M.D.

9:40 Results of the Surgical Treatment of Peptic Ulcer at the San Juan City Hospital.
Ricardo Arredondo, M.D.
Discussion: José Noya-Benítez, M.D.

10:00 The Heart and its Relation to Thyroid Dysfunction.
Roberto Rodríguez, M.D.
Discussion: Agustín M. de Andino, M.D.

10:20 Intermission

10:40 Aminophylline Overdosage in Children.
Arnaldo García, M.D.
Discussion: Etervina Figueroa, M.D.

11:00 Clinicopathological Conference.
Jerome W. Conn, M.D.
Enrique Koppisch, M.D.

JUEVES 8 DE DICIEMBRE

Asociación Médica
2:00 p.m.

Presidirá el Dr. Ramón A. Sifre

2:00 p.m. Tri-Demensional Drawing for the Blind. (With Demonstrations). Mr. George Wally

2:20 Ultra Sound Therapy - Some personal Observations. Herman J. Flax, M.D.

2:40 Congenital Familial Telangiectasia. A. A. Cintrón-Rivera, M.D. and José A. de Jesús, M.D.

3:00 Bilharzial Heart Disease in Puerto Rico: Cor Pulmonale due to Schistosomal Pulmonary Endarteritis. Ernesto J. Marchand, M.D., Roberto Rodríguez, M.D., Raúl Marcial-Rojas, M.D. and Gerardo Polanco, M.D.

3:20 On the Evaluation of Dyspnea in the Patient with Pulmonary Tuberculosis. Hilda García de la Noceda, M.D. and José M. Torres, M.D.

3:40 The Midline Cervical Disc Syndrome. Nathan Rifkinson, M.D.

4:00 The Problem of Cerebral Aneurysm. Bernard J. Alpers, M.D.

4:45 Induction and Stimulation of Labor. William F. Finn, M.D.

(La noche del JUEVES estará libre para actividades sociales de grupos)

VIERNES 9 DE DICIEMBRE

Hospital San Patricio
8:30 a.m.

Presidirá el Dr. José Chaves-Estrada

8:30 a.m. Ward Rounds, all services.

9:30 Electrolyte Changes in Congestive Heart Failure. Elí A. Ramírez, M.D

9:50 Follow-up Report on Carcinoma of the Stomach. Luis A. Passalacqua, M.D., and Héctor M. Nadal, M.D.

10:10 Thrombocytopenic Purpura due to Fuadin. Julio V. Rivera, M.D., H. Rodríguez, M.D. and E. Pérez-Santiago, M.D.

10:30 Follow-up Report on Carcinoma of the Lung.
Alfred L. Axtmayer, M.D. and
José A. Sárraga, M.D.

10:50 Responsibility of the Radiologist in the Diagnosis of Chest Diseases.
Laszlo Ehrlich, M.D.

11:10 Clinicopathological Conference.
F. Hernández-Morales, M.D. and
Félix M. Reyes, M.D.

1:00 p.m. Lunch.

VIERNES 9 DE DICIEMBRE
Asociación Médica
2:30 p.m.
Presidirá el Dr. Eugenio M. de Hostos

2:30 p.m. Symposium: Breast Cancer
Raúl A. Marcial-Rojas, M.D., Moderator
Luis A. Vallecillo, M.D.
Ramón E. Llobet, M.D.
Pablo Luis Morales, M.D.
Víctor A. Marcial, M.D.

3:30 Multiple Cancers. Pablo Luis Morales, M.D.

3:50 Cáncer del Esófago.
Jaime L. Costas-Durieux, M.D.

4:10 Cancer of the Colon in Puerto Rico.
Raúl A. Marcial-Rojas, M.D.,
and Rubén Medina, B.S.

4:30 The Surgical Treatment of Cancer of the Esophagus and Cardia with an Appraisal of Long Term Results.
John H. Garlock, M.D.

5:15 Studies on the Pathogenesis of Cirrhosis of the Liver.
Arnold R. Rich, M.D.

VIERNES 9 DE DICIEMBRE
Asociación Médica
8:15 p.m.
Presidirá el Dr. José A. de Jesús

8:15 p.m. The Cineplastic Method for Upper Extremity Amputation. (Illustrated by motion picture)
Rufus H. Alldredge, M.D.

9:00 The Diagnosis and Treatment of sciatica.
Bernard J. Alpers, M.D.

9:45 Differential Diagnosis and Treatment of Spontaneous Hypoglycemia.
Jerome W. Conn, M.D.

SABADO 10 DE DICIEMBRE

Asociación Médica

9:30 a.m.

Presidirá el Dr. Elí A. Ramírez

9:30 a.m. Milk Allergy in Children. Joseph Aponte, M.D.

9:50 Mucoviscidosis. E. S. Colón-Rivera, M.D. and Rafael Blasini, M.D.

10:10 The management of Special Problems in the Chemotherapy of Tuberculosis in Infants and Children. José E. Sifontes, M.D.

10:30 Hepatitis: Analysis of 175 cases. Carlos E. Bertrán, M.D., Ramón A. Sifre, M.D., Calixto A. Romero, M.D. and J. A. de Jesús, M.D.

10:50 Studies on the Nature and Effects of Hypersensitivity Arnold R. Rich, M.D.

11:35 Management of Abnormal Bleeding at Term. William F. Finn, M.D.

SABADO 10 DE DICIEMBRE

Asociación Médica

2:00 p.m.

Presidirá el Dr. A. M. de Andino

2:00 p.m. Studies on Insulin Zinc Modifications. Manuel E. Paniagua, M.D., A. M. de Andino, M.D., Emilio Ramírez Kohl, M.D. and José H. Rivera, M.D.

2:20 Determination of Blood Volume in Healthy Puerto Ricans using Chromium 51. Roberto Busó, M.D.

2:40 Fibrinolytic Purpuras E. Pérez-Santiago, M.D. and C. E. Butterworth, M.D.

3:00 The Intravenous Cholangiogram in the Diagnosis of Biliary Tract Disease. F. Hernández-Morales, M.D. and Pablo Luis Morales, M.D.

3:20 Low Back Pain with and without Sciatica.
Rufus H. Alldredge, M.D.

4:05 Surgical Therapy of Cancer of the Colon and Rectum.
John H. Garlock, M.D.

4:50 Toma de posesión de la nueva directiva.

EXHIBICIONES CIENTIFICAS

Las siguientes exhibiciones científicas estarán instaladas en el salón que ocupa la Biblioteca en la segunda planta del edificio:

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"Social Workers Help Heart Patients"
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Labor que realiza el Negociado de Niños Lisiados.
Departamento de Salud

"Congenital Heart Disease"
Egidio S. Colón Rivera, M.D.

"The Diagnostic Value of Laminography"
Laszlo Ehrlich, M.D.

El problema del cáncer en Puerto Rico.
Liga Puertorriqueña Contra el Cáncer

Diagnóstico diferencial de la tuberculosis.
Clínica Antillas

El diagnóstico citológico.
Enrique Koppisch, M.D.

Tumors in Puerto Rico

Raúl A. Marcial, M.D.
Gustavo Ramírez de Arellano, M.D.
Antonio Puras, M.T.
Rafael Ramírez, B.S.

Tri-Dimensional Drawing for the Blind
George Wally

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Clínica de la Concepción; Madrid

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Los cursos se dividen en cuatro tipos: según su duración e intensidad:

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10—Dermatología

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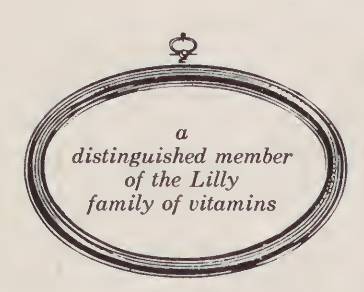
ASAMBLEA ANUAL

Diciembre 6-10, 1955

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BOLETIN

DE LA

ASOCIACION MEDICA DE PUERTO RICO

VOL. 47 DICIEMBRE, 1955 NO. 12

Entered as second class matter, January 21, 1931 at the Post Office at San Juan, Puerto Rico, under the act of August 24, 1912.

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FECHA DE PUBLICACION:

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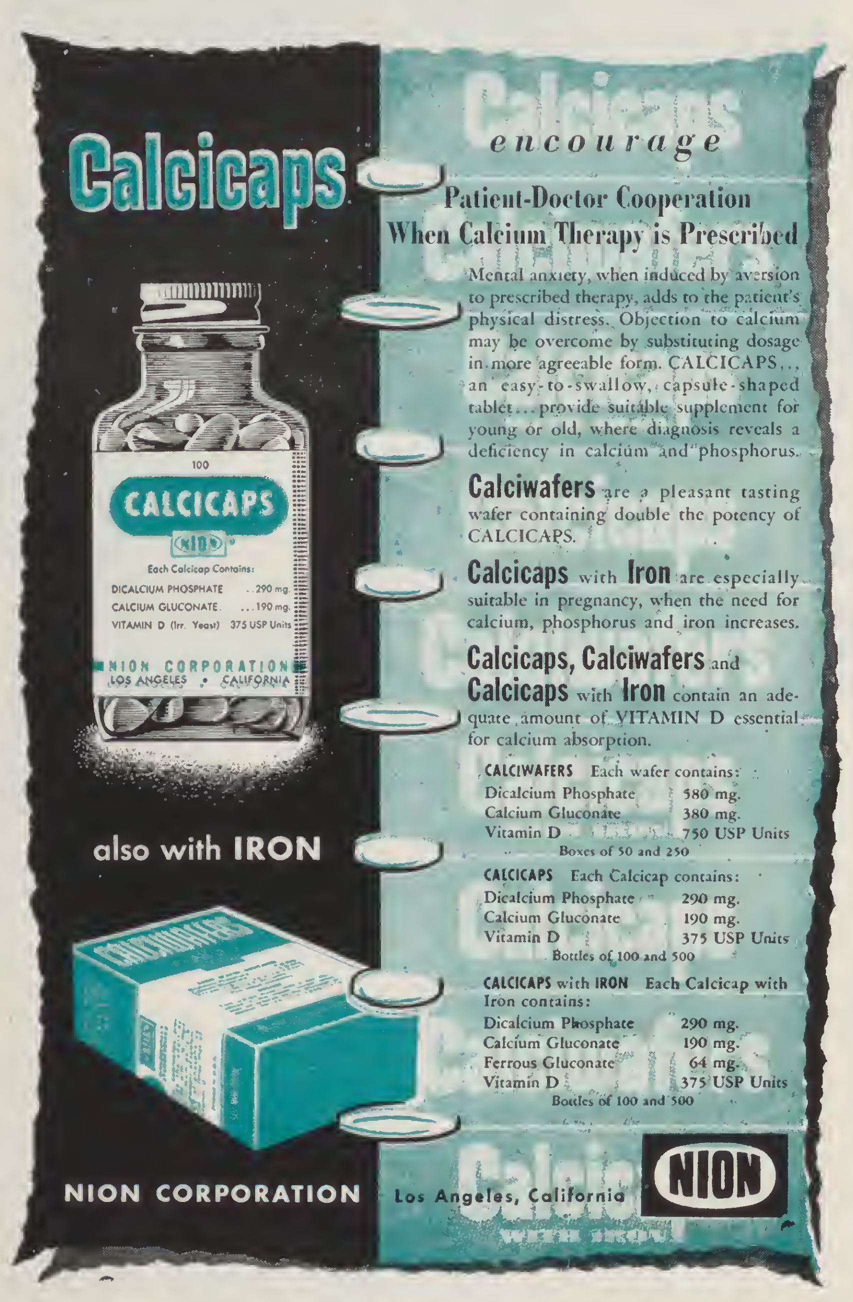


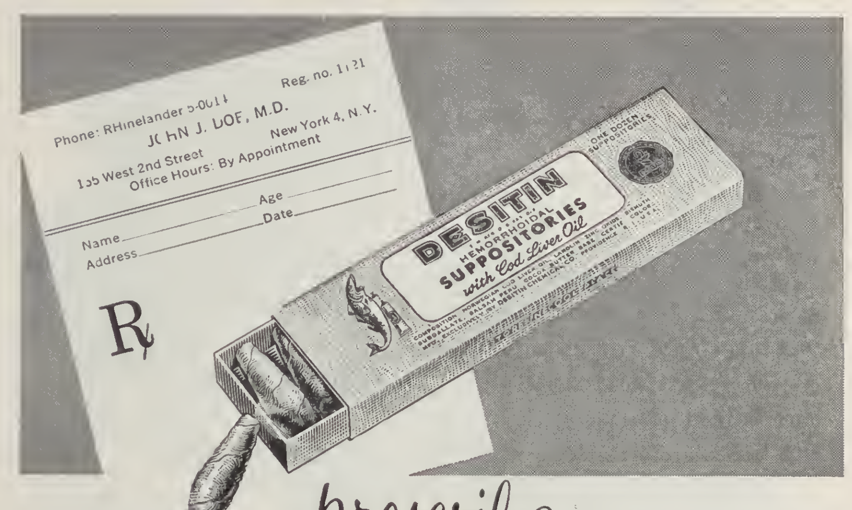
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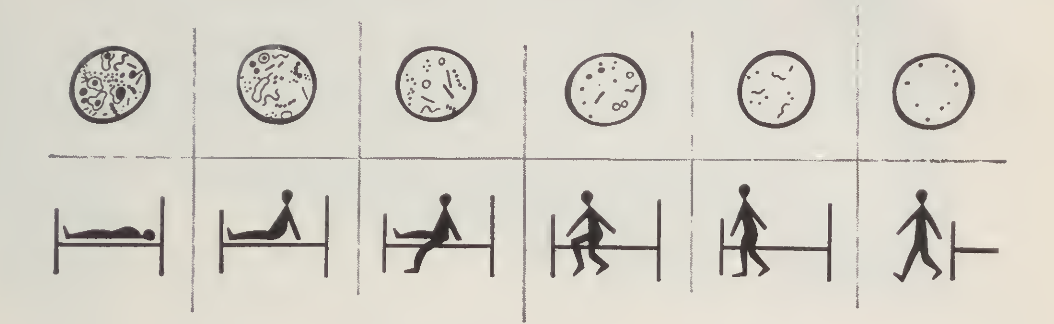
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BOLETIN

DE LA ASOCIACION MEDICA DE PUERTO RICO

VOL. 47 DICIEMBRE, 1955 No. 12

MUCOVISCIDOSIS

*EGIDIO S. COLÓN RIVERA, M.D., F.A.A.P.**
*RAFAEL BLASINI, M.D.***

Mucoviscidosis which is also called Cystic Fibrosis of the Pancreas, is a generalized disease affecting primarily the mucous secreting glands; and it is the effect that this has, on the pancreas and the respiratory system, that brings the patient to the doctor.

It has been during the past decade that Mucoviscidosis has emerged as a very distinct and important clinical entity. Till a few years ago we were convinced that the disease did not exist here in Puerto Rico, since then we are becoming conscious that not only is it present here, but that probably it is more frequent than we suspect.

In a review of 1,578 consecutive autopsies, which were performed at the School of Tropical Medicine during the years 1940 to 1953; five cases were found in which the pathologic diagnosis of Cystic Fibrosis of the Pancreas had been made. The first case was a 5 months old infant admitted to the University Hospital in July 1944 with a diagnosis of Athrepsia, chronic Enterocolitis and possible Malaria. The second case was a 5 months old infant admitted to the Presbyterian Hospital in April 1946 with a diagnosis of Bronchopneumonia and Diarrhea. Cystic Fibrosis of the Pancreas was mentioned in the differential diagnosis. The third case was a 4 months old infant admitted to the San Juan City Hospital in February 1949 with a diagnosis of malnutrition. The fourth case was an 8 months old infant admitted to the San Juan City Hospital on March 1952 with a diagnosis of Bronchial Asthma. And the 5th case was a 27 days old premature admitted to the San Juan City Hospital on January 1953 with a diagnosis of Gastroenteritis and Bronchopneumonia. It is interesting to note that in the 4th case the possibility of Mucoviscidosis had been considered and the patient had had three negative Shwachman's tests, but

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** Chief of Pediatrics, Ponce District Hospital.

studies of the duodenal fluid could not be carried out. The admission diagnosis in these few patients can give you an idea about the many conditions with which Mucoviscidosis has been confused.

On November 8, 1953 a 5 lbs. premature was born in the Nursery of the San Juan City Hospital. Since birth the premature presented abdominal distention accompanied with vomiting. A diagnosis of intestinal obstruction was made and the patient was operated. At operation he was found to be a case of Meconium Ileus which in turn meant that this was also a case of Mucoviscidosis.

Since then we have been able to study two cases of this condition at the San Juan City Hospital and as they are, as far as we know, the first cases that have been diagnosed and studied completely, here in Puerto Rico, we are presenting them with the hope that it will make us more conscious of this disease.

One is an 8 months old girl and the other is a 5 yrs. old child both of whom came to the Hospital on March 1955 with the chief complaint of chronic respiratory infections and chronic diarrhea. The family history in the 8 months old infant was strongly suggestive of this disease. Of the 8 children that the mother had had, 3 had died before the age of one year with symptoms similar to the ones shown by our patient. The other four siblings gave a history of repeated respiratory infections. The other child has 2 brothers who also have frequent respiratory infections but no marked Gastrointestinal disturbances. This shows what has been known for some time, that Mucoviscidosis is a familial disease probably transmitted as a recessive characteristic; which explain why you usually have more than one member of the family with the disease. It is also important to point out that when one case has been diagnosed in the family the other siblings should be screened for the disease, because even if they don't show the full-blown picture, some might show evidence of what has been called "Partial Pancreatic Insufficiency".

Both cases had a similar history with the symptoms being more marked in the 8 months infant. In both, the first evidence of the disease were respiratory infections, which would affect the upper and the lower respiratory tract. In the older child a diagnosis of Bronchial Asthma had been made in a previous occasion; in the younger, there was a persistent dry cough which had not responded to a quite intensive therapy. Later, both developed a chronic type of diarrhea with foul, bulky stools and with occasional episodes of acute diarrhea.

In the study of these cases the first thing that was very striking was the degree of failure in growth. The 8 month infant had a weight of 12 lbs. and a height of 24½ inches. The 5 years old child had a weight of 27½ lbs and a height of 39 inches in their

measurements. Both had prominent abdomens with the wasting more marked in the extremities, with flaccid muscles and prominent gluteal folds. They looked unhappy, crying very easily showing evidence of the change in mental disposition, chiefly irritability, that has been described in this disease.

Staphylococcus aureus was cultured from the nasopharynx of the 8 months infant. The culture from the older child yielded Alpha and Beta Hemolytic Streptococci. It has been pointed out that Staphylococcus aureus is almost constantly present in the nasopharynx of these children and this is so much so, that their demonstration is considered as added evidence for the diagnosis of Mucoviscidosis.

The Volmer Patch Test and the PPD were negative in both patients as were the serology, urinalysis, stool cultures and stool sample for eosinophyles. The oldest patient showed Ascaris, Trichuris and Hookworm in the stools, for which he received treatment. An oral Glucose Tolerance curve was done and both patients showed a normal curve which is what is seen in over 80% of these patients, in contradistinction to cases of Idiopathic Coeliac Disease where it is usually flattened.

Through the facilities of the Army Research Laboratory a Vitamin A absorption test was done to demonstrate the defect in fat absorption. A blood sample was taken to determine the fasting Blood level of Vitamin A. Then they were given about 0.1 cc per lb. of body weight of Oleum Percomorphum, orally, and 5 hrs. and 7 hrs. later, other blood samples were taken to find out the rise in vitamin A level in the blood. The normal fasting level of vitamin A is between 30 and 60 units, and after the test dose it usually rises about 130 Units above the previous level, usually reaching its peak in about 5 hrs. The normal level of carotene is about 130 micrograms and its rise follows closely the Vitamin A Curve. Both of our patients showed abnormally low levels. (See Table 1). This test is not as useful as others in the diagnosis of Mucoviscidosis because there are several conditions of non pancreatic origin which also affect the digestion and absorption of fats. But usually these other conditions don't show as marked a depression in the absorption of fats as in Mucoviscidosis. Added evidence of the presence of Mucoviscidosis can be obtained by showing that there is a significant rise in the Vitamin A level if Pancreatin is given together with the Oleum Percomorphum or if a water soluble preparation is used instead, but we were unable to do this.

The demonstration of trypsin in the stools, which has been called "Shwachman's Test", is a useful screening procedure in cases where Mucoviscidosis is suspected. It is done by placing

Table 1

VITAMIN A ABSORPTION TEST

8 Months girl	Vitamin A	Carotene
Fasting	0	0
5 hrs.	25	0
7 hrs.	40	0
5 Yrs. Old Child	**Vitamin A**	**Carotene**
Fasting	25	85
5 hrs.	25	85
7 hrs.	40	85

diluted stool specimens on an unfixed X-Ray film, using a control and incubating the specimens. If trypsin is present, a clear area will appear in the film showing that the gelatin has been digested. Three negative Tests will usually confirm your diagnosis, but there can be both false positives and false negatives. We did this procedure on three different occasions in both patients and both gave 4 + values, and in one case in which the dilution was carried up to 1:120 it still showed a 1+ value. We explained these findings on the basis that both patients had been receiving antibiotic therapy for some time and that there were present proteolytic bacteria in the feces.

The next procedure that was carried out and the one which is the most important in the diagnosis of Mucoviscidosis, was duodenal intubation. This is very useful because you can measure both the amount of tryptic activity, and the viscosity of the fluid. It has been pointed out that in Mucoviscidosis the duodenal fluid is abnormally viscous and scanty. In both our cases we felt that the viscosity was increased but it could not be actually measured because of lack of adequate facilities. Trypsin was absent from the duodenal fluid in the 5 yrs. old child and definitely diminished, as seen in Mucoviscidosis, in the 8 months infant. (See Table 2)

There are many other laboratory procedures that can also be done in these patients, like chylomicron counts, the Gelatin Absorption Test, the levels of the different enzymes in the duodenal fluid and the determination of Sodium Chloride in the thermal sweat and saliva. These last two tests will probably become very helpful in the screening of patients suspected of having Mucoviscidosis.

From the roentgenological point of view, these patients sometimes present sufficiently characteristic findings so that the radiologist can suggest the diagnosis from the examination of films

Table 2

TRYPSIN AND VISCOSITY IN THE DUODENAL FLUID

	UNDILUTED					1/10 DILUTION	1/100 DILUTION			VISCOSITY (Ostwald Viscosimeter)
	volume ml.					volume ml.	volume ml.			Time in Seconds
	1.0	0.8	0.6	0.4	0.2	1 cc.	1.0	0.5	0.25	
Normal	+ +	+	+	+	+	+	+	0 or +	0	Average range 70 - 120
Mucoviscidosis	0 or +	0 or +	0 or +	0 or +	0	0	0			Over 180
8 Month Infant Alkaline sample	+	+	0			0	0			Increased
Acid sample	+	+	0			0	0			Increased
5 yr. old child	0	0	0			0	0			Increased

of the chest. In the early cases the findings are those of emphysema combined with areas of atelectasis and peribronchial infiltration. Later, superimposed infections will show patchy areas of pneumonic consolidation. A chronic bronchopneumonic process should always raise the question of Mucoviscidosis. The 8 months old infant showed a bilateral bronchopneumonia in the admission X-Ray and when this was repeated 3 weeks later she still presented more or less the same radiologic picture. The 5 yrs. old child had an increase in the bronchial markings.

During their course in the hospital, both patients were treated with antibiotics and received, as part of their therapy; an adequate diet, water soluble vitamins, and Pancreatin. When their condition improved, they were discharged to be followed up in the Out Patient department.

SUMMARY

Two cases of Mucoviscidosis have been presented and it is hoped that increased interest in this disease will result in its more frequent and earlier diagnosis.

Acknowledgment:

The data on the autopsies was collected by Mr. Rubén Medina, 4th year Medical Student of the School of Medicine of the University of Puerto Rico.

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UN CASO INTERESANTE DE OBSTRUCCIÓN DE LAS VÍAS BILIARES*

*ANTONIO H. SUSONI, M.D.***

Este caso fué presentado en la reunión anual de la Asociación Médica de Arecibo del 1953 con el siguiente título "Estenosis del conducto común post colecistectomía", pero como la etiología de la obstrucción coledociona resultó ser de otra naturaleza a la que supusimos, volvemos a presentarlo porque ello reafirma lo adecuado de nuestra conducta y criterio en cuanto a la actitud que adoptamos con este paciente, así como también reafirma las conclusiones a que llegamos y que fueron expresadas en esa Asamblea.

Dijimos entonces lo siguiente:

"La cirugía de las vías biliares puede ser extremadamente sencilla o por el contrario, presentar grandes dificultades técnicas. Estas dificultades pueden ser debidas a la inflamación aguda o crónica de la vesícula biliar y secundariamente de los órganos vecinos, lo que acarrea cambios patológicos de tal naturaleza que hace sumamente difícil la disección e identificación de las diversas estructuras hepatobiliares. Por otra parte, muy frecuentemente existen en esa región anomalías fundamentales. Por cuyas razones se hace imperativo en este tipo de cirugía el hacer una disección minuciosa de manera de poder identificar fuera de toda duda cada uno de los órganos envueltos en la mencionada región. De lo contrario, se expone el cirujano a incurrir en graves errores técnicos; siendo uno de los más serios, la ligadura parcial o completa del conducto común. Lahey considera que esta complicación es uno de los problemas más difíciles con que puede confrontarse un cirujano en lo que respecta a establecer ulteriormente el drenaje biliar al intestino con resultados satisfactorios y permamentes.

"Lahey y Peytek quienes han tenido gran experiencia en la reconstrucción de las vías biliares por estenosis post-operatorias, creen que estos accidentes ocurren cuando hay hemorragia de la arteria cística o hepática; confusión en la anatomía de la unión del cístico, común y conductos hepáticos o anomalías de los conductos y vasos sanguíneos causando dudas en sus relaciones anatómicas; podemos añadir a esto el hacer tracción excesiva en el cístico en el momento de aplicarle la ligadura o pinzas lo que podría acarrear interesar en dicha ligadura parte o todo el conducto común, produciendo una obstrucción parcial o completa del mismo, según la magnitud del segmento interesado."

---

* Trabajo presentado en la Asamblea Anual del Distrito de Arecibo, el 10 de octubre de 1954.

** Director Médico y Cirujano, Hospital Dr. Susoni, Arecibo, P. R.

"Una de las causas más frecuentes de hemorragias es la laceración o ruptura de la arteria cística. Esto se debe al hecho de que esta arteria tiene su origen en la hepática derecha y es más corta que el conducto cístico. Es fácil de comprender por qué una tracción exagerada del cuello vesicular puede producir rotura de la arteria cística produciéndose una hemorragia que irremisiblemente obscurecerá el campo operatorio. Si no se tiene presente la maniobra sencilla de comprimir el ligamento hepatoduodenal por donde pasa la arteria hepática lo más probable es que el cirujano recurra en su desesperación a la aplicación de pinzas a ciegas con la esperanza de conseguir la hemostasis; poniendo así desde luego en gran riesgo los canales hepáticos."

"Voy a presentarles un caso sumamente interesante cuya estenosis de los conductos biliares evidentemente no obedece a ninguna de las causas mencionadas."

"Se trata de paciente J.L.C., caso 4218— blanco, de 52 años de edad, quien a fines del año 1952 comenzó a desarrollar dolor en el cuadrante superior derecho y al mismo tiempo, las escleras se pusieron amarillas, orina rojiza, heces blanquecinas y picor en todo el cuerpo. Fué hospitalizado en el Hospital de Distrito de Arecibo en donde se le hizo un estudio de la vesícula biliar, no visualizando ésta. Se trató médicamente y mejoró algo y fué dado de alta. En febrero 12 de 1953 se exacerbaron todos los síntomas. El estado general del paciente empeoró y desarrolló ictericia intensa con gran prurito sin gran dolor y con ligera febrícula. Se hospitalizó en la Clínica Dr. Susoni. El índice ictérico fué de 97.4; Van den Bergh directo fuertemente positivo y la reacción indirecta 14.8; cefalin colesterol Positivo+. En febrero 24, 1953 el índice ictérico subió a 156 y el día 3 de marzo bajó a 100. El paciente fué dado de alta a petición del mismo.

"Fué readmitido el 13 de marzo de 1953 en la Clínica Dr. Susoni y operado el 17 del mismo por el Dr. Valdivieso. Se le encontró una vesícula distendida y engrosada. El cístico fibrótico y el conducto común no distendido y permeable, se exploró y se dejó un tubo en T. Los dilatadores pasaron al duodeno. No había colelitiasis ni coledocolitiasis." El diagnóstico patológico del Dr. Koppisch fué el siguiente. Colecistitis sub-aguda y crónica y dice que las paredes estaban gruesas y rígidas. El día 25 fué dado de alta habiendo mejorado su estado general y cedido notablemente la ictericia."

"Pocos días después el paciente comenzó a drenar bilis por el labo del tubo y a sentir dolor en el epigastrio. Sin embargo, el índice ictérico había bajado a 22. Subsiguientemente tuvo crisis de escalofríos y fiebre y el tubo aparentemente se había salido del conducto común, estaba como dos pulgadas más afuera de donde

se fijó y la irrigación con solución salina se salía por los lados del mismo. Se removió el tubo pero continuó drenando bilis por la fístula."

"En junio 1ro., fué reingresado por tercera vez en la Clínica Dr. Susoni por continuar con la fístula y haber vuelto a deteriorarse su condición general. El índice ictérico era de 46. Jugo gástrico para bilis fué negativo. El B.S.P. a los 30 minutos era 29%. Hanger Pos. +++. Albúmina 4.50 grm. Glob. 1.95 gm. Orina: Bilis positivo; Urobilinógeno neg., Bilirrubina positivo +; Van den Bergh directo e indirecto positivo; Protrombina normal; Bilis en la excreta negativa."

"Con estos datos le hicimos un diagnóstico de ictericia obstructiva de las vías biliares.

"Fué nuestra opinión que la ictericia por obstrucción que desarrolló este paciente después de la intervención no se debió a las causas usuales que antes mencionamos; es decir, traumatismo o ligadura accidental de las vías biliares externas puesto que estas fueron debidamente identificadas como lo demostró el hecho de que el tubo en T fué introducido en el conducto común y por donde drenó bilis por un cierto tiempo. La posibilidad de una piedra impactada en la ampolla de Vater también podía descartarse de una manera razonable puesto que el conducto fué explorado y el sondeo de la ampolla de Vater fué posible; un tumor de la cabeza del páncreas, las alzas y las bajas del índice ictérico, la no visualización de la vesícula en el colecistograma, ni la obstrucción en la ampolla de Vater favorecían esa posibilidad. Luego entonces nos queda una sola causa que podía explicar razonablemente la obstrucción y que era la siguiente: Al salirse el tubo en T parcialmente de su sitio, ésto es, del conducto común, obstruyó la salida de las bilis a través del lúmen de dicho tubo y por ende al duodeno, por cuya razón el jugo biliar buscó salida por el punto de menos resistencia que es alrededor del tubo de Kehrs, no sin antes formarse un pozo de bilir más o menos grande o pequeño alrededor de las vías hepáticas externas. Todos sabemos que se ha escrito con frecuencia sobre el efecto fibrosante de la bilis, que posiblemente en este caso fué la causa de la estenosis de los conductos hepáticos y común y ésto causante a su vez de que la fístula externa se estableciera con carácter permanente.

Es obvio y fuera de toda discusión que el procedimiento ideal de reparación en los casos de estenosis de las vías biliares, es eliminar el trozo obliterado y hacer una anastomosis término-terminal si la parte distal y próxima estuviesen utilizables o también una coledocoduodenostomía o coledocoyeyunostomía y si las vías biliares externas estuvieren inutilizables una hepato-yeyunostomía o hepato-duodenostomía por el método de Catell.

Pero los procedimientos arriba mencionados no siempre son posibles realizarlos y mucho menos con gran probabilidad de éxito, dado el caso de que el plastrón fibroso pueda ser de tal naturaleza que la disección de esa región fuese técnicamente imposible. En relación a esta cirugía, Lahey en su última edición "Práctica quirúrgica de la Clínica Lahey" se expresa en estos términos. "Estas operaciones han agotado todos nuestros recursos de ingeniosidad quirúrgica tratando de afrontarlo y muy frecuentemente nos ha defraudado con sus resultados. En otro párrafo del mismo artículo menciona la transplantación de la fístula externa al intestino con el único propósito de descartarla como intervención de valor práctico. Uno de estos casos inaccesibles a los procedimientos descritos y que presencié en un Hospital en E. U. me sirvió de experiencia para el caso que les presento hoy. Uno de los más notables cirujanos de esa Institución en un caso similar al nuestro, después de largas horas de disección no pudo realizar la operación que tenía en mente, y habiendo destruído la fístula externa, el único recurso que tuvo fué el dejar un tubo de drenaje para que esta se realizase espontáneamente. Ignoro si el paciente sobrevivió a este último recurso. La razón que aducen en la Clínica Lahey para descartar la utilidad del trasplante de la fístula externa es que ésta al ser transplantada al intestino, se estenosa en corto período de tiempo. Dan el ejemplo, de que una fístula biliar interna espontánea, tarde o temprano, se cierra debido a que la retracción del tejido fibroso que constituye sus paredes es mayor que la presión que ejerce la secreción de la bilis que es lo único que permite mantener la fístula abierta.

El caso que les presento lo intervinimos en junio 12 de 1953, o sea, 3 meses y 1 semana después de la colecistectomía. Al abrir el vientre nos encontramos que tenía todas las características de inoperabilidad, en el sentido de poder disectar el proceso inflamatorio de carácter fibroso e inflamatorio en donde todas las estructuras de esa región estaban involucradas. Insistimos en la disección hasta donde creímos que ya era temeridad en continuarla. Pero durante todo ese proceso respetamos la fístula externa por todos los medios posibles, la cual habíamos desprendido de la pared abdominal y la utilizamos en la forma siguiente:

Se nos ocurrió durante el acto operatorio, con el fin de evitar la estenosis del conducto fistuloso (1ro.) remover de sus paredes la mayor cantidad posible de tejido fibroso, consiguiendo dejarla tan fina como el espesor de una vena de calibre mediano. Conseguimos disectarla hasta la cara inferior del hígado en una extensión de más de dos pulgadas. (2do.) Con el mismo fin, al implantarla en el yeyuno, en vez de hacer una incisión axial en la pared del intestino, removimos un redondel de un diámetro un poco ma-

yor que el de la fístula, tal y como hacemos en las gastrectomías totales para evitar la constricción del estoma esófago-yeyunal. (3ro.) Añadimos otra variante a la técnica de implantación de este tipo de fístula que es el hacer una yeyuno-yenunostomía para evitar en lo más posible el reflujo alimenticio en la fístula. El enfermo tuvo un post-operatorio tranquilo y una convalescencia sin complicaciones, reduciéndose rápidamente la ictericia."

El paciente vino nuevamente a consulta a la Clínica Dr. Susoni el 24 de febrero de 1954 alegando que se había sentido bien hasta enero de ese mismo año o sea, 10 meses después del trasplante fistuloso, cuando comenzó a tener náuseas y vómitos alimenticios con molestias en el hipocondrio derecho con irradiación al bajo vientre. En febrero 25 se le hicieron los siguientes análisis: Urobilinógeno positivo en la orina; índice ictérico 10.4 (normal), azúcar 108; Hanger 3+. Se le hizo estudio gástrico el cual reveló lo siguiente: Estómago muy dilatado. El barium no pasaba más allá de la segunda porción del duodeno, media hora después una cantidad mínima había pasado al intestino delgado. El radiólogo añade que con excepción de la obstrucción marcada en el asa descendente del duodeno no había evidencia de patología gástrica achacando estos cambios a posibles cicatrizaciones secundarias a las operaciones que se le había hecho, y su recomendación fué laparatomía. Fué operado el 26 del mismo mes y nos encontramos con una masa tumoral que invadía toda la cabeza del páncreas e interesaba el duodeno, había muy poca evidencia de proceso inflamatorio. Había nódulos tumorales en el hígado, y como hallazgo más notable la fístula permanecía intacta, sus paredes completamente blandas y con un diámetro más o menos idéntico al que tenía cuando hicimos su trasplante. Se tomó un biopsia del hígado y se hizo una gastro-yeyunostomía anterior.

El paciente tuvo un post-operatorio satisfactorio y fué dado de alta el día 7 de mayo o sea, 11 días después de intervenido. Murió 4 meses después en su casa sin síntomas de obstrucción duodenal ni biliar.

Informe patológico: Adeno-carcinoma metastásico al hígado del páncreas.

Queda demostrado sin lugar a dudas que si en aquella ocasión en que trasplantamos la fístula hubiésemos temeraria e insensatamente insistido en llegar hasta las vías hepáticas para realizar algunos de los procedimientos antes mencionados, nos hubiésemos encontrado desamparados puesto que es evidente que para la época en que se hizo el trasplante, las vías biliares estaban ya totalmente inutilizables debido a la invasión tumoral.

Para resumir podemos decir que se trataba de un caso de obstrucción de las vías biliares que aún cuando no se hizo el diagnóstico etiológico correcto se consideró sensatamente inoperable en lo que se refiere a restablecer el flujo biliar por alguno de los métodos mencionados, y que se procedió a realizarlo con lo único que convenía en este caso específico.

Y en conclusión podemos decir lo siguiente:

Que hemos demostrado que el trasplante de una fístula externa a alguna parte del sistema intestinal superior, duodeno o yeyuno, no es un método inútil y si de posible gran valor en ciertas circunstancias como la antes expresada; además, que un año es un tiempo razonable para poner a prueba la permanencia funcional de una fístula trasplantada tanto más que para esa época nada evidenció que se hubiese estado estableciendo estenosis. Por otra parte es bueno añadir que apesar de que el proceso tumoral había avanzado notablemente, la inflamación de esa región afectada. por el contrario, había cedido visiblemente y que de no haberse tratado de un cáncer, el paciente hubiese estado en condiciones muy favorables para restablecer la continuidad de las vías biliares si así se hubiese deseado.

Creemos además que el éxito que obtuvimos con este paciente mediante un método que no goza del apoyo del eminente grupo de cirujanos de la Clínica Lahey, quienes quizás son los más experimentados en esta materia, pueda deberse a las variantes que le introdujimos al procedimiento con el propósito de evitar la tendencia a la estenosis de estas fístulas biliares.

FUNDACIÓN DEL DEPARTAMENTO DE SALUD PÚBLICA Y MEDICINA PREVENTIVA DE LA ESCUELA DE MEDICINA

*PABLO MORALES OTERO, M.D., F.A.C.P.**

Este Departamento se inauguró oficialmente al comienzo del curso académico de 1940-1941 teniendo como objeto inmediato impartir enseñanza de salud pública a un número determinado de estudiantes. Entonces existía la antigua Escuela de Medicina Tropical y desempeñaba el cargo de Comisionado de Sanidad el fenecido compañero Doctor Eduardo Garrido Morales. La Escuela contaba con la decidida cooperación del Doctor Garrido y el Departamento de Sanidad que él dirigía contribuía al sostenimiento del Departamento con fondos procedentes de las asignaciones federales que éste recibía.

El primer Director del Departamento fué el Doctor Albert V. Hardy, doctorado en salud pública y eminente epidemiólogo muy conocido por sus estudios en Brucelosis y las disenterías.

Seis meses se dedicaron a la organización de los cursos, selección de personal, obtención de material para enseñanza y equipar los laboratorios necesarios. Se habilitaron los laboratorios de bacteriología, parasitología y química, y aulas para bioestadísticas y trabajo de planos, laboratorios adicionales a los que ya contaba la vieja Escuela. Los gastos que se ocasionaron con estas obras fueron sufragados con fondos federales e insulares.

En el mes de noviembre de ese año llegó a la Escuela el profesor J. M. Henderson para hacerse cargo de las clases de ingeniería sanitaria, en el mes de enero, la señora Johanna Schwarte para encargarse del entrenamiento de las enfermeras de salud pública y en el mes de febrero, el Doctor Myron E. Wegman para la enseñanza de higiene maternal e infantil.

El doctor James A. Doull, de Western Reserve University, desempeñó el cargo de profesor visitante por un período de seis meses, dictando conferencias sobre epidemiología e iniciando la labor de investigación de dicho Departamento con un estudio sobre diarrea y enteritis, causa importante de muerte en nuestra isla. El doctor Morton Kramer tuvo a cargo la enseñanza de estadísticas, mientras al mismo tiempo desempeñaba un puesto a cargo de la División de Estadísticas del Departamento de Sanidad. Se nombraron además otros técnicos sanitarios de la isla con experiencia profesional para desempeñar funciones pedagógicas.

Ese año se entrenaron cuatro grupos de estudiantes, entre los cuales figuraban 10 enfermeras de salud pública seleccionadas en-

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* Presidente de la Comisión de Salud y Beneficencia, Cámara de Representantes de Puerto Rico.

tre las superintendentes y enfermeras graduadas del Departamento de Sanidad; 10 ingenieros sanitarios; y 13 ayudantes de laboratorio, todos con el grado de Bachillerato en Ciencias procedentes de la Universidad de Puerto Rico y del Instituto Politécnico de San Germán.

El personal facultativo de otros departamentos ya establecidos en la Escuela se encargaba de explicar bacteriología, química de la nutrición y parasitología. El Colegio de Educación de la Universidad de Puerto Rico y una Unidad Modelo de Salud Pública establecida en Río Piedras se encargaban de impartir instrucción en todos los aspectos del programa de enseñanza.

Durante ese año los Doctores Walter Clarke y M. K. Keyes, pertenecientes a la Sociedad Americana de Higiene Social, dieron un cursillo intensivo sobre los distintos aspectos de las enfermedades infecciosas génitourinarias. El Doctor A. Ashley Weech, del Hospital de Niños de la ciudad de New York, profesó un curso graduado de pediatría. A estos cursos asistieron 15 médicos, todos ellos funcionarios del Departamento de Sanidad.

La labor de investigación del Departamento empezó al mismo tiempo que la labor docente. El doctor Hardy y el Doctor Kramer hicieron un estudio con miras a evaluar la garantía que ofrecen en este país las causas de muerte tal como aparecen consignadas en los certificados de defunción. Empezaron el estudio de las diarreas infantiles prestando atención especial a un brote de disentería bacilar causada por el **B. New Castle.** El Doctor James Watt, del Servicio de Salud Pública de los Estados Unidos, estudió con preferencia los tipos de disentería bacilar que prevalecen en la isla y la epidemiología de la enfermedad. El profesor Henderson planeó un estudio especial del problema de la malaria en las zonas urbanas de este país y con la ayuda de sus discípulos planeó una investigación sanitaria de un caserío en la jurisdicción de Trujillo Alto, cuyo plan de investigaciones fué multigrafiado mientras otra investigación semejante se llevaría a cabo en el distrito municipal de Río Piedras. Auxiliado por el personal de los Departamentos de Bacteriología y Medicina Interna el profesor Wegman investigó el tratamiento de la disentería bacilar con sulfaguanidina y ciertos factores etiológicos y epidemiológicos en relación con las diarreas de la Infancia. El Doctor Kramer en unión al pediatra puertorriqueño Doctor Fernández Marchante llevó a cabo una investigación de la alimentación infantil en el país y su relación con la mortalidad en los primeros años de vida. El epidemiólogo Doctor James A. Doull, con la ayuda de sus estudiantes llevó a cabo una investigación referente a la tuberculosis en la urbanización rural de Saint Just.

Los cursos organizados fueron preparados para oficiales mé-

dicos de sanidad con el objeto de que pudieran obtener su diploma con Licenciatura (Master) en Salud Pública, para enfermeras de salud pública con el título en esta especialidad, para ingenieros graduados que deseaban especializarse y recibir el título de "Master" en Ingeniería Sanitaria y para Bachilleres en Ciencia con el objeto de obtener un certificado acreditado en Tecnología Médica.

Durante este período se llevó a cabo un cursillo intensivo de Pediatría y Obstetricia en el Hospital de Distrito de Bayamón y otro sobre "Abastecimientos de Agua" preparado especialmente para los ingenieros sanitarios del Departamento de Sanidad.

Aparte de los cursos regulares, la especialista en nutrición señora Rosa M. de Rodríguez, de la Universidad de Puerto Rico, dirigió una serie de lecciones prácticas para el adiestramiento del personal del Departamento de Sanidad.

Finalmente, los miembros del Departamento de Salud Pública contribuyeron a organizar y practicaron activamente sobre problemas médico-sociales referentes a Puerto Rico que fueron provistos en la Escuela de Trabajo Social de la Universidad de Puerto Rico.

El Doctor Hardy, que dirigía el Departamento, renunció para marcharse a los Estados Unidos ocupando su puesto como Director del Departamento el Doctor J. M. Henderson.

En el curso del 1942-1943 se matricularon por primera vez estudiantes con el objeto de hacer trabajos especiales: el Doctor Carlos Calero, de Manta, Ecuador; el Doctor José de J. Alvarez, de Santo Domingo; y la señorita María Dolores Fernández, del mismo país. Durante el mes de febrero la señorita Aimeé Wilcox, del Servicio de Salud Pública de los Estados Unidos, explicó un curso superior sobre microscopía en relación con el paludismo al cual asistieron 52 alumnos; y el Doctor Honorato De Castro, catedrático de Ciencias Matemáticas de la Universidad Central de Madrid y astrónomo de aquel observatorio, profesó un curso de conferencias sobre "Teoría de Mínimos Cuadrados" con especial aplicación a las bioestadísticas. El Doctor Henderson fué ascendido a profesor de ingeniería sanitaria de la Universidad de Columbia en New York, y en la dirección del Departamento fué nombrado el Doctor Guillermo Arbona, puertorriqueño meritísimo que ha venido desempeñando esa labor hasta el presente.

En este año se gradúan por primera vez de Maestros en Salud Pública (Master in Public Health) los Doctores E. Martínez Rivera, Luis A. Sánchez y Rafael A. Timotheé.

En el año 1914 se nombró al señor José Rivera León, profesor auxiliar de ingeniería sanitaria, a la señorita Edna S. MacKinnon, profesora auxiliar de enfermería sanitaria, a la señorita Celia Guzmán como ayudante de la misma disciplina, al señor Orlando Bonilla como ayudante de laboratorio, al señor Nelson Biaggi

profesor agregado de ciencias sanitarias y al profesor Earl B. Phelps profesor visitante de ciencias sanitarias. Además de los 72 estudiantes que asistieron a los cursos regulares del Departamento se profesaron cursos especiales de ingeniería sanitaria, al cual asistieron 24 estudiantes, otros para inspectores de sanidad con el objeto de entrenar un grupo de 20 estudiantes, 10 de los cuales procedían de la República de Haití enviados por la Oficina de Coordinación de Asuntos Interamericanos. Más tarde se llevó a cabo un tercer curso para 19 estudiantes, 9 procedentes de la República Dominicana.

En el año 1945, previa consulta con la Junta Especial de Síndicos de la Escuela de Medicina Tropical, se decidió cambiar el nombre del Departamento a Departamento de Higiene. Durante este año vinieron a Puerto Rico 8 estudiantes enviados por el Instituto de Asuntos Interamericanos, 3 eran médicos y 5 ingenieros, y asistieron a los cursos regulares 108 estudiantes los cuales en su mayoría procedían del Departamento de Sanidad.

En el mismo año la Legislatura de Puerto Rico incluyó en su Plan de Seis Años una asignación de $240,000 para la construcción de un nuevo edificio para alojar el Departamento de Higiene. El reputado arquitecto Don Rafael Carmoega fué encargado del diseño y preparación de planos para el nuevo edificio. El Departamento quedó instalado en el nuevo edificio a principios del año 1949.

En el 1946 la matrícula a dicho Departamento fué como sigue: 4 estudiantes de Brazil, 1 de Colombia, 3 de la República Dominicana, 5 de la República de Haití, 2 de Venezuela, y 73 procedentes de la isla. Este año se dictó un curso práctico sobre el control de malaria. Los estudiantes que asistieron al mismo estuvieron la mayor parte del tiempo fuera de la Escuela, en el mismo campo de operaciones, llevando a cabo una encuesta preliminar en Loíza Aldea y después realizando una campaña de saneamiento antimalárico en cooperación con el Departamento de Sanidad y el Servicio de Salud Pública de los Estados Unidos en distintos puntos de la isla. Entre los estudiantes matriculados en este curso asistieron al mismo 5 oficiales del ejército americano, encargados de la tarea antimalárica en los puestos militares donde estaban destacados. Se profesó además, un curso sobre métodos de laboratorio de ingeniería sanitaria en el que figuraban diversas materias de enseñanza: procedimientos químicos y bacteriológicos de aguas potables y aguas impuras, exámenes de leche y utensilios y enseres utilizados en la preparación de alimentos.

Se continuó la investigación científica en cooperación con otros Departamentos de la Escuela llevando a cabo un estudio de la distribución de los casos de tifus exantemático acaecidos en el

recinto de nuestra Capital. Se continuó el estudio epidemiológico de las diarreas y enteritis llevando a cabo estudios bacteriológicos e inmunológicos en los asilados del Hospital de Psiquiatría. Se inició una nueva tarea de investigación que comprendía el estudio de las condiciones sanitarias de las aguas de la Bahía de San Juan, el Caño de Martín Peña y la Laguna San José. Este estudio resultó de gran interés para la Autoridad de Acueductos y Alcantarillados de Puerto Rico.

En el próximo año, además de continuar la labor docente, se dió mayor importancia a la de investigación estudiando una vacuna preparada con un microorganismo de **Flexner IV** en 90 enfermos shigelósicos practicándose semanalmente el examen coprológico de todos ellos para comprobar el índice de infección entre los vacunados y los no vacunados. En colaboración con el Departamento de Zoología Médica se emprendió un estudio para determinar el efecto que producen distintos preparados de cloro en el tratamiento químico de las aguas de alcantarillados sobre las larvas y los huevecillos de la bilharzia.

Estudióse la frecuencia con que aparecen bacilos tuberculosos en la leche de vacas con reacción positiva a la tuberculina. El personal encargado de la Sección de Estadísticas del Departamento colaboró con el Centro de Estudios Sociales de la Universidad de Puerto Rico en un estudio de nuestro problema de población emprendido con la Oficina de Investigaciones Demográficas de la Universidad de Princeton.

El Doctor Marcelino Pascua, miembro del Departamento de Bioestadísticas de la Escuela de Salud Pública de la Universidad de Johns Hopkins, profesó un curso sobre estadísticas demográficas y métodos estadísticos y el Doctor James S. Simmons, decano del Colegio de Salud Pública de la Universidad de Harvard, concurrió invitado como orador a la Asamblea Anual de la Asociación de Salud Pública de Puerto Rico.

Se prosiguió el estudio de la diarrea entre los enfermos del Hospital de Psiquiatría determinando el organismo causante de las infecciones en el mismo. De 7,000 muestras examinadas 228 resultaron positivas, la mayor parte perteneciente al tipo **Flexner.** Sin embargo, también aparecieron infecciones con la **Sh. schmitzi** y **Sh. alkalescens.** Se observaba cierta variación estacional en la incidencia de la enfermedad, la cual disminuía en frecuencia durante el mes de diciembre llegando al punto mínimo en mayo. Empezaban a aumentar los casos en el mes de junio y alcanzaban el máximo en el mes de septiembre. Se encontró que algunos casos infectados con determinado tipo de **Sh. Flexner** no sufrían reinfección, mientras que otros enfermos padeciendo de infección por otro tipo de microorganismo sufrían reinfección con frecuencia,

lo cual demostraba que algunos tipos de **Flexner** confieren una inmunidad más duradera que otros.

A solicitud de la Autoridad de Acueductos y Alcantarillados se llevó a cabo un estudio del grado de polución del Río Mata de Plátano en las montañas de Luquillo y a solicitud del Departamento de Sanidad se llevó a cabo una extensa investigación de la contaminación de las aguas del Río Puerto Nuevo y un estudio del funcionamiento del alcantarillado de Bayamón. Una fábrica de cerveza de la localidad pidió así mismo, que se practicase una investigación para determinar la demanda bioquímica de oxígeno para las aguas de desperdicio industrial.

En el mes de febrero de 1947 se inició una campaña sistemática para el control de la esquistosomiasis en un sitio denominado "Las Peñas", una zona suburbana del barrio de Sabana Llana de Río Piedras, con 600 habitantes que residían en 110 domicilios en los cuales el parasitismo esquistosomiásico alcanzaba el 40% de la población. Se pusieron en práctica las siguientes medidas de control: (a) tratamiento de los sujetos parasitados, (b) construcción de letrinas, (c) erradicación del caracol que sirve de huésped intermediario del parásito, y (d) instrucción popular sobre los medios de evitar la parasitosis. Este experimento se llevó a cabo por varios años, habiendo sido publicados sus resultados en revistas científicas.

Lo escrito anteriormente les dará a ustedes una idea clara de lo que es el Departamento, que hace poco reconoció la Asociación Americana de Escuelas de Salud Pública. No quiero terminar sin dar entero crédito a los Doctores E. Garrido Morales, Bachman y Hardy, que concibieron la idea de su establecimiento y colaboraron en su fundación, y felicitar calurosamente a mis compañeros que componen la Facultad de dicho Departamento, al profesor Guillermo Arbona y al actual Secretario de Salud, Doctor Juan A. Pons, quienes merecen crédito por su laudable esfuerzo.

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Los directores de la Asociación Médica asisten a un agasajo ofrecido por el presidente de la A.M.A., Dr. Elmer Hess, durante su reciente visita a Puerto Rico. De izquierda a derecha: Dr. F. Hernández Morales, Dr. C. José Ferraioli, Dr. Ricardo F. Fernández. Dr. Jaime F. Pou, Dr. Luis R. Guzmán López, Dr. F. Sánchez Castaño y Dr. Guillermo Picó. Al frente, en cuclillas, el Dr. Elmer Hess.

DISCURSO PRESIDENCIAL*

Invitados de honor, compañeros, damas y caballeros:

No cabe duda de que la medicina en Puerto Rico ha hecho grandes avances en los últimos años. Hemos seguido el ritmo de progreso de la ciencia médica en el mundo entero y especialmente en EE. UU., ya que nuestra Asociación Médica está afiliada a la Asociación Médica Americana y nuestro pueblo se desarrolla en unión política con el pueblo americano.

En 1940 teníamos en la isla sólo 509 médicos para atender una población de 1,800,000 habitantes o sea, un médico para cada 3,500 personas; hoy contamos con 1,419 médicos para una población de 2,240,000, que nos da un médico por cada 1,600 personas, que es una proporción muy buena de acuerdo con la economía del país.

Tenemos una escuela de medicina de primera categoría, capaz de suplir suficientes médicos bien preparados para cubrir las necesidades futuras del país. Hay además unos 500 jóvenes puertorriqueños estudiando medicina en EE. UU. y en el exterior, muchos de los cuales vendrán a sumarse a nuestras filas en los próximos 5 años.

Los hospitales privados y gubernamentales se han multiplicado y han modernizado su equipo, y hoy están, muchos de éstos, a la altura de los mejores de América. Puerto Rico tiene, relativamente, mayor número de hospitales privados que muchos otros países.

Todos estos adelantos han traído consigo mayores servicios médicos y de más alta calidad. El promedio de vida del puertorriqueño que era sólo de 46 años en 1940, en la actualidad alcanza 64 años y la mortalidad general es menor de 8 por cada 1000 habitantes por año, cifra que compara favorablemente con la de los EE. UU.

La mejor atención médica es parte del gran movimiento de la humanidad hacia una vida mejor como son las mejores viviendas, mejores salarios y mejores condiciones de trabajo.

Naturalmente estos grandes adelantos en la atención médica y hospitalaria han traído consigo un aumento considerable en el costo de los servicios médicos. Y este aumento ha traído la necesidad de nuevos métodos de distribución de estos servicios.

Y es precisamente en esta fase de la práctica de la medicina, una de las más importantes por cierto, porque toca más de cerca las necesidades del pueblo, donde nos hemos quedado rezagados.

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* Pronunciado por el Dr. Ricardo F. Fernández, presidente de la Asociación Médica de Puerto Rico, en la sesión inaugural de la quincuagésima-segunda Asamblea Anual, Diciembre 6, 1955.

En Estados Unidos se está librando una gran batalla entre la libre empresa y la regimentación de los servicios médicos y en esta batalla la clase médica americana ha luchado gallardamente del lado de la libre empresa y ha triunfado.

¿Por qué luchó la Asociación Médica Americana contra la Regimentación?

Porque la regimentación no admite la libre competencia y desvirtúa las relaciones entre el paciente y el médico, principios sobre los cuales ha estado basada la práctica ética de nuestra profesión desde sus comienzos, y que han probado a través de los siglos que son indispensables a la buena práctica de la medicina.

¿Y qué hizo la clase médica Americana para conseguir este triunfo?

Idear y estructurar planes de seguro médico, que conservando el sagrado principio de la libre selección del médico por el paciente, brindaron al pueblo mejores servicios médicos al menor costo posible.

Actualmente hay en Estados Unidos más de cien millones de personas cubiertas por algún tipo de seguro médico o de hospitalización. Esto cubre aproximadamente dos terceras partes de la población.

¿Y nosotros, qué hemos hecho?

Hemos hecho algo, no hay duda de eso, pues en Puerto Rico hay unas 300,000 personas cubiertas por los planes de seguro médico, como la Cruz Azul, el Auxilio Mutuo, el plan de la Asociación de Maestros y otros planes particulares. Pero estos planes sólo cubren una octava parte de nuestra población y algunos de ellos dejan mucho que desear en cuanto a la calidad de sus servicios y en la limitación de los riesgos que cubren.

El pueblo demanda más y mejores servicios médicos, y nosotros estamos en la obligación de prestárselos.

Ahora estamos nosotros frente al mismo problema que tuvieron nuestros compañeros del Norte. Altos funcionarios de nuestro Gobierno se han manifestado públicamente en favor del seguro médico compulsorio, y nosotros que sabemos que este sistema sólo produciría menos y peores servicios para el pueblo interponiendo entre el paciente y su médico la ineficiencia burocrática, tenemos que combatirlo del mismo modo que lo hizo el pueblo norteamericano y su clase médica.

Hay que tener en cuenta que nuestro problema local es mucho mayor que el de los EE.UU. porque allá el 90% de las familias tienen suficientes ingresos para cubrir el costo de los servicios médicos si este costo se distribuye en pequeños plazos a través de un sistema de seguro médico.

Aquí el promedio de ingresos por familia es mucho más bajo

que en EE.UU. y tendremos necesariamente que ajustarnos a esta realidad para poder resolver nuestro problema. Y aquí cabe la pregunta que sé está en la mente de todos ustedes: ¿Puede el público de Puerto Rico pagar estos servicios? Estamos seguros que sí. Nuestro ilustre gobernador ha dicho recientemente, y cito sus palabras: "nuestras metas son de un ingreso mínimo, no promedio —de $1500 al año por familia para alguna fecha antes del 1960, y $2000 para el 1960 o para no mucho después de 1960. Consideramos que se ha logrado ese mínimo cuando el 90% de las familias puertorriqueñas estén sobre él."

Se estima que toda familia con un ingreso de $1800 anuales o más puede disponer de unos dólares al mes para pagar un seguro médico que le proteja en caso de enfermedad. Sabemos que en muchos casos como en el de nuestro propio Gobierno, el patrono ayuda a sus empleados a cubrir el costo del seguro médico. Nuestra meta es un plan que pueda cubrir en un futuro cercano el 80% de nuestra población.

Uno de los efectos más importantes de un plan de seguro médico de esta naturaleza es el de emancipar a miles de personas de su condición de indigencia médica. Al puertorriqueño de pocos recursos le es desagradable y a veces cree denigrante tener que depender de la caridad pública o privada para tratar sus males. Participando en un plan voluntario de seguro médico se sentirá orgulloso de no necesitar de esta caridad.

Comprendemos que la estructuración y administración de un plan de esta magnitud es una tarea hercúlea que demandará todas nuestras fuerzas y nuestro ingenio y requerirá el sacrificio de muchos intereses personales en pro del bien común.

Pero tenemos convicción plena de que la clase médica puertorriqueña tiene en su seno suficientes profesionales con excelente preparación para poder proveer a nuestro pueblo de servicios médicos tan buenos como los mejores que puedan obtenerse en cualquier otro país en el mundo, y por eso hemos comenzado a estructurar un plan para ofrecerle al pueblo estos servicios que ellos están demandando con legítimo derecho.

La Cruz Azul de Puerto Rico ha aceptado todos nuestros requisitos para elaborar un plan conjunto y está ansiosa de unir sus recursos a los nuestros para llevarlo a feliz término. Pero todavía falta mucho por hacer. Para triunfar en un plan de esta naturaleza se necesita la cooperación de los médicos que proveen los servicios y del público que los usa y que los paga. Estamos dispuestos a considerar cuantas sugestiones tengan a bien hacernos las agencias gubernamentales, las uniones obreras y las entidades privadas que al igual que nosotros deben estar interesados en que

se ofrezcan a nuestro pueblo servicios médicos de la más alta calidad al menor costo posible.

La clase médica tiene que asumir el liderato en esta cruzada, y tenemos que redoblar nuestros esfuerzos para evitar la regimentación gubernamental de los servicios médicos que traería desprestigio y sometimiento a nuestra profesión y desgracia y sufrimiento a nuestro pueblo.

Ricardo F. Fernández, M.D.
Presidente

INFORME DEL PRESIDENTE

AÑO 1955

Estimados compañeros:

Al iniciar nuestra gestión administrativa en diciembre del año pasado nos propusimos realizar el mayor esfuerzo para cumplir a cabalidad la misión que ustedes tuvieron a bien hacernos. Decidimos, sin embargo, dar mayor énfasis a los siguientes aspectos del programa trazado:

1— Ampliación del Edificio
2— Campaña de Socios
3— Programa Científico
4— Relaciones Públicas
5— Aumento en los Sueldos de los Médicos
6— Boletín Médico

Puedo asegurarles que si en alguno de estos asuntos no hemos logrado alcanzar nuestro objetivo no ha sido por que nuestro interés haya decaído, y sí por las dificultades que el mismo conlleva.

En relación con el aumento de sueldo que gestionábamos para los médicos que trabajan para el gobierno estatal agotamos todos nuestros recursos. Sostuvimos entrevistas con el honorable Secretario de Salud y con el Director de la Oficina de Personal. Ambos ejecutivos nos prometieron toda su ayuda; pero dificultades de índole presupuestal impidieron que pudieran completarse este año los trámites necesarios para hacer efectivo el aumento solicitado.

Tengo la certeza, sin embargo, que tanto el Dr. Pons como el Sr. Torres Braschi están en buena disposición y debemos seguir laborando en pro de este aumento. Para esto se necesitará una cooperación más efectiva de los médicos envueltos que la que hemos conseguido hasta ahora.

Campaña de Socios

Aunque en este asunto no alcanzamos la meta de 100 nuevos socios que nos habíamos impuesto, resulta bastante halagador el notar que durante el año ingresaron a nuestra agrupación 58 socios regulares y 4 afiliados.

Es de conocimiento general que el éxito de las gestiones de cualquier agrupación como la nuestra depende principalmente del número de personas que esté respaldándola. En la época actual cuenta mucho la cantidad; no es suficiente la calidad. Ello nos lo

prueba el hecho de que las uniones obreras muy pocas veces pierden alguna de sus demandas, y más que por la justificación de éstas se debe ello al respaldo casi total de sus integrantes.

No pretendemos, ni siquiera remotamente, que la profesión médica se una con el fin deliberado de hacer demandas injustificadas; pero sí nos gustaría ver una clase médica unida y poderosa que pueda luchar con éxito en la defensa de su legítimo derecho a mantenerse libre de intromisiones e imposiciones ajenas, a fin de poder rendir a la población servicios médicos de la más alta calidad.

Es deber de cada uno de nosotros abogar porque se hagan socios todos aquellos compañeros que permanecen alejados de nuestras filas y que consideremos dignos de entrar al grupo.

Sabemos que muchos de ellos la única excusa que ofrecen para no formar parte de la Asociación es la de que ésta no hace nada por ellos. Los que así piensen, realmente no son buenos candidatos para pertenecer a nuestra organización. Vamos a atraer en primer término los que digan ¿qué podemos hacer nosotros por la Asociación? Los que vienen a un grupo pensando sólo en el beneficio que éste les puede proporcionar no vacilarán en abandonarnos cuando tengamos que hacer frente a alguna situación que ellos estimen pueda perjudicar sus intereses.

Es verdad que toda Asociación tiene el deber ineludible de velar por el bienestar de sus socios, ya que son éstos los que hacen posible su existencia; pero es también cierto que el socio tiene la responsabilidad de servir a su asociación y anteponer el interés y bienestar de ésta al suyo propio. Cuantos han servido en la dirección de la Asociación desde su organización en septiembre de 1902, así lo han hecho. No vemos razón alguna para que todos los socios no estén en igual disposición. Sólo cuando todos adoptemos esa actitud lograremos hacer de la Asociación una entidad respetada y querida por nuestro pueblo y muy particularmente por los que dirigen nuestro gobierno.

Boletín Médico

Es con verdadera satisfacción que anotamos el hecho de que por primera vez en varios años se ha logrado poner al día la publicación de nuestro Boletín.

Este año, por disposición reglamentaria hubo necesidad de nombrar para presidir la Junta Editora a un nuevo compañero; pero al ser llamado el doctor Vallés al ejército, volvió a asumir la dirección el anterior presidente, doctor Jiménez López, bajo cuya hábil dirección y la cooperación eficiente de los demás miembros de la Junta Editora se ha puesto al día el Boletín.

La Madison Pharmaceutical Laboratories representada en Puerto Rico por el Lcdo. Adolfo Legrand, ha establecido tres premios para ser otorgados a los mejores trabajos que se publiquen en el Boletín. Estos premios constan de una placa y cheque por $100 para el primero; cheque de $50.00 para el segundo, y $25.00 para el tercero. Este año la Junta Editora seleccionó los siguientes trabajos:

Primer Premio

GUILLAIN-BARRE SYNDROME - PRESENT CONCEPTS AND REPORT OF 14 CASES

Héctor F. Rodríguez, M.D., Ernesto C. Martínez, M.D., y Luis R. Guzmán-López, M.D.

Publicado en la edición de enero de 1955.

Segundo Premio

ISONIAZID BY THE INTRATHECAL ROUTE IN THE THERAPY OF TUBERCULOUS MENINGITIS

José E. Sifontes, M.D., Carmen L. Berio, M.D., Katherine R. Rivera, M.D. and Julieta Grana, M.D.

Publicado en la edición de enero de 1955.

Tercer Premio

CARCINOMA OF THE STOMACH - SEVEN-YEAR REVIEW

John F. Sanabria, M.D.

Publicado en la edición de julio de 1955.

Esperamos que estos premios sirvan de estímulo para que nuestros compañeros den al Boletín sus publicaciones de interés, descontinuando la práctica de enviarlos a revistas americanas.

Deseamos reiterar por medio de estas líneas nuestro agradecimiento sincero a los Laboratorios Farmacéuticos Madison por la ayuda que nos brindan.

Relaciones Públicas

Otro de los asuntos objeto de nuestro interés ha sido el referente a relaciones públicas.

Como es de vuestro conocimiento desde el domingo 19 de junio, hemos venido trasmitiendo a través de la radicemisora WKAQ, todos los domingos a las 12:00 del día y bajo los auspicios de Ma-

dison Pharmaceutical Laboratories, un programa radial con el nombre de "Los Médicos Informan".

El comité de Relaciones Públicas, bajo la entusiasta dirección del Dr. José A. de Jesús, y con la cooperación del Sr. René Poitevín, quien actúa como locutor y moderador, ha venido presentando a varios compañeros en la discusión de temas de interés general para la comunidad.

Este programa ha tenido gran aceptación en la radio-audiencia, y esperamos que el mismo pueda seguirse llevando a cabo.

A petición de los auspiciadores nos proponemos hacer una encuestra entre médicos para verificar qué acogida tiene este programa entre la clase médica.

Durante el año hicimos innumerables gestiones para iniciar un programa de televisión y logramos conseguir a los Laboratorios Pfizer como auspiciadores; pero nos dimos cuenta de las serias dificultades que un programa de esta naturaleza conlleva, y desistimos de la idea para no desviar nuestra atención de otros problemas que a nuestro juicio eran de más urgencia.

Hemos discutido ampliamente en el seno de la directiva la necesidad y conveniencia de crear una división de relaciones públicas para ocuparse más bien de tener al tanto a la directiva de todos los proyectos de carácter médico que se presenten en la Legislatura y de ayudarnos a combatir los que consideremos perjudiciales y a defender aquellos que sean beneficiosos. La persona a cargo de esta división deberá también asesorar a la directiva en cuestiones públicas de carácter controversial. Hasta la fecha se han considerado varios candidatos; pero en la opinión de la mayoría uno de éstos reúne las mejores cualificaciones. Es un abogado prominente y persona muy influyente en los círculos gubernamentales. Nos proponemos entrevistarnos con él en el curso de esta asamblea, y mientras tanto, nos hemos anticipado a crear un nuevo cargo en el presupuesto con una remuneración de $3,000 anuales.

Ampliación del Edificio

Esta presidencia tiene un interés muy marcado en conseguir poner el domicilio de nuestra Asociación al nivel de la clase a la cual alberga.

Posiblemente todos ustedes, al igual que los demás miembros de la Asociación se sentirán hastiados de mis contínuas exhortaciones para que cooperen en esta causa. Sinceramente no nos arrepentimos de nuestra insistencia, puesto que con ello hemos logrado levantar el interés de un gran número de compañeros.

Actualmente tenemos en caja $37,000.00 incluyendo el beneficio del sorteo del carro, que como ustedes saben, se llevará a efecto por segunda vez durante el mes en curso. Este nos ha dejado un

beneficio de $6,000 y esperamos produzca algo más con el cobro de los boletos pendientes y los que se puedan vender antes del nuevo sorteo.

En nuestro empeño de poder empezar esta obra a principios de enero próximo ya hemos sometido los planos y especificaciones a la consideración de tres ingenieros contratistas amigos, y sus proposiciones serán consideradas oportunamente por el Comité de Edificio.

Esta presidencia está altamente agradecida a los compañeros que integran el Comité de Edificio, muy especialmente a su presidente, Dr. Basilio Dávila y al Dr. Freddie González, quienes han demostrado verdadera disposición e interés para que pueda realizarse este proyecto. Son igualmente acreedores a nuestra gratitud todos los colegas que nos han animado en nuestro empeño a través de sus generosos donativos y de su desinteresada ayuda en la colocación de boletos para el sorteo de este año.

Tribunal Examinador de Médicos

Con fecha 28 de diciembre de 1954 enviamos una carta al honorable Gobernador sometiéndole las siguientes ternas para cubrir las vacantes ocurridas en el Tribunal el 27 de dicho mes:

Para sustituir al doctor Barbosa:

Dr. Luis R. Guzmán López
Dr. Federico Hernández Morales
Dr. J. R. González Giusti

Para sustituir al doctor Pérez:

Dr. E. Colón Yordán
Dr. Luis F. Sala
Dr. J. Rodríguez Olmo

Para sustituir al doctor Pons:

Dr. Luis A. Sanjurjo
Dr. Manuel E. Paniagua
Dr. José M. Torres Gómez

No logramos sin embargo, que se tomara acción sobre estas vacantes, toda vez que en la pasada sesión legislativa el Senador Rivera Colón presentó un proyecto de ley para eliminar las juntas examinadoras y el Gobernador no deseó hacer nuevos nombramientos hasta tanto se supiera en qué paraba dicha legislación.

Este proyecto recibió una franca oposición de nuestra parte y el periódico El Mundo en su edición del 6 de mayo publicó un jui-

cioso editorial oponiéndose a la aprobación de una medida tan drástica.

Más recientemente vino a nuestro poder un esquema de proyecto cuya intención es quitarle a nuestra Asociación el derecho de proponer ternas al honorable Gobernador para miembros del Tribunal. Dicho embrión de proyecto lee como sigue:

L E Y

Para enmendar el Artículo 1ro. de la Ley Número 22 de Abril de 1931 enmendada subsiguientemente.

Decrétase por la Asamblea Legislativa de Puerto Rico:

Sección 1. Por la presente se enmienda el Artículo 1ro. de la Ley Número 22 aprobada el 22 de abril de 1931 y enmendada subsiguientemente para que lea como sigue:

Artículo 1.—Al empezar a regir esta ley, el Gobernador de Puerto Rico, por y con el Consejo y consentimiento del Senado de Puerto Rico, (a propuesta de las sociedades o asociaciones médicas, debidamente inscritas en el Departamento de Estado de Puerto Rico, como sociedades o asociaciones para fines no pecuniarios) nombrará por un término de cuatro años, un Tribunal Examinador de Médicos. compuesto de siete médicos, con no menos de cinco años de ejercicio de la profesión en el Estado Libre Asociado cada uno, y de acuerdo con las siguientes disposiciones; Disponiéndose, que no más de tres de estos médicos serán residentes de la ciudad de San Juan.

Las sociedades o asociaciones médicas debidamente inscritas en el Departamento de Estado de Puerto Rico como sociedades o asociaciones para fines no pecuniarios podrán someter al Gobernador una lista de candidatos que reúnan los requisitos para ser designados miembros de la Junta Examinadora. El Gobernador podrá utilizar esta lista en la selección de dichos miembros.

Sección 2. Esta ley empezará a regir inmediatamente después de su aprobación.

Esta misma semana tuvimos una cordial entrevista con el honorable Gobernador, en la cual estuvieron presentes el Dr. Jaime F. Pou, presidente electo, y el Dr. A. Oliveras Guerra, presidente del Tribunal Examinador de Médicos de Puerto Rico. Durante este cambio de impresiones el Sr. Gobernador nos explicó los motivos por los cuales él no había nombrado los nuevos miembros de la Junta; nos explicó que la legislación propuesta por el Senador Rivera Colón aparentemente había perdido el favor legislativo y nos ofreció que procedería a extender los nombramientos pendientes en el curso de los próximos días.

Nuestra Asociación ha mantenido relaciones muy cordiales con el Tribunal Examinador y nuestros asesores están actualmente interviniendo como "amicus curiae" en unos pleitos que llevan contra el Tribunal varios de los médicos extranjeros que pretenden se les extienda licencia permanente sin el requisito de someterse a los exámenes regulares de reválida.

En caso de que la decisión sea desfavorable al Tribunal Examinador de Médicos ya hemos solicitado personalmente del Subsecretario de Justicia que se recurra en apelación al Tribunal Supremo.

Asociación Médica Americana

Todos ustedes saben de la acogida franca y cordial de que fueron objeto los doctores Pou y Picó, durante la visita que dispensaron a las oficinas de la Asociación Médica Americana en Chicago y Washington en gestiones oficiales en relación con legislación pendiente de consideración en nuestro país.

No cabe duda que las conexiones hechas por los doctores Pou y Picó habrán de sernos de gran beneficio en el futuro. Es necesario que se estimulen esas relaciones de amistad de manera que podamos seguir utilizando al máximo la gran influencia que tiene la Asociación Médica Americana en las esferas gubernamentales americanas.

Nececitamos demostrar a la Asociación Médica Americana nuestra gratitud por las atenciones dispensadas a nuestros delegados, y se nos ocurre que la mejor forma de expresar dicho agradecimiento es consiguiendo que un mayor número de médicos puertorriqueños se hagan miembros de esta gran organización.

Este año tenemos entre nosotros al Dr. Hess, buen amigo de nuestra clase médica y actual presidente de la Asociación Médica Americana. Demostrémosle a él nuestro aprecio y hagamos su permanencia en Puerto Rico una de grata recordación.

Auxilio Médico Mutuo

Actualmente hay una disposición en el reglamento del Auxilio Médico Mutuo para que los médicos que ingresen después del período de gracia concedido paguen además de la cuota regular de $50.00, el siguiente recargo:

1. Médicos que ingresen después del primer año de haber revalidado y que sean menores de 30 años de edad $2.00
2. Médicos de 31 a 40 años de edad 7.00
3. Médicos de 41 a 50 años de edad 17.00
4. Médicos de 51 a 60 años de edad 32.00

Esta disposición no se ha seguido al pie de la letra, ya que se interpretó que dicho recargo se cobraría solamente el primer año. Por otro lado el mismo resultaría muy fuerte si se cobrara anualmente.

Así pues, interpretando el sentir de mis compañeros de directiva vamos a proponer que dicha escala de recargos sea enmendada en la forma siguiente:

Póliza A —	Hasta los 35 años de edad el médico que ingrese a la Asociación pagará la cuota regular, que actualmente es de --------	$50.00
Póliza B —	De 36 a 40 años pagará un recargo anual de ------------------------------	5.00
Póliza C —	De 41 a 50 años pagará un recargo anual de ---------------------------	10.00

Médicos que deseen ingresar a la Asociación después de haber cumplido los 50 años no se podrán acoger al plan de Auxilio Médico Mútuo.

Comité de Credenciales.

El Comité de Credenciales de la Asociación, bajo la presidencia del Dr. Antonio Navas, ha venido realizando una eficiente labor en relación con las solicitudes de ingreso.

En su última reunión el Comité decidió no considerar más solicitudes para la categoría de miembros afiliados hasta tanto se enmiende el reglamento para determinar el tiempo durante el cual podrán permanecer éstos en dicha categoría y recomendó a la directiva se presentara en esta reunión la correspondiente enmienda al reglamento.

Accediendo a la solicitud del Comité de Credenciales deseamos proponer la siguiente enmienda al inciso C de la Sección Primera del Artículo I del Reglamento:

Para que donde dice:

> "La condición de "miembro afiliado" cesará seis meses después que el candidato haya recibido su notificación de haber aprobado los exámenes de reválida, y deberá entonces hacer su solicitud para miembro activo."

se le agregue el siguiente Disponiéndose:

> "Disponiéndose, que bajo ningún concepto un médico podrá permanecer en esta categoría por un período mayor de cinco años."

Médicos jubilados

Como es de conocimiento general, los médicos que ahora trabajan para el Departamento de Salud son obligados a retirarse al cumplir los 65 años. Muchos de estos compañeros, al llegar a esta edad, sólo cuentan con la pensión que les dará el Gobierno, limitándose por lo tanto considerablemente sus ingresos.

Ya nuestra directiva ha recibido la solicitud de uno de estos compañeros para que se les exima del pago de la cuota.

Con el fin de remediar esta situación, que en el futuro habrá de repetirse con bastante frecuencia, nos permitimos proponer la siguiente enmienda a la Sección Segunda del Artículo I del Reglamento.

Agregar al final de la sección el siguiente párrafo:

"Todo médico que haya pertenecido a la Asociación Médica de Puerto Rico por un período de 30 años y cumplido 65 años de edad, podrá ser eximido del pago de la parte de la cuota correspondiente a la Asociación mediante solicitud que deberá ser aprobada por la junta directiva."

Cruz Azul y Escudo Azul

Todos ustedes están al tanto de las gestiones que ha venido haciendo la directiva de la Asociación conjuntamente con el Comité de Escudo azul y Cruz Azul de la Cámara en relación con el programa de Escudo Azul.

Vamos a transcribir para conocimiento de ustedes la última correspondencia cursada sobre este asunto entre la Asociación y la Cruz Azul de Puerto Rico.

3 de octubre de 1955

Honorable Junta de Directores
Cruz Azul de Puerto Rico
Santurce, P. R.

Señores:

La Asociación Médica de Puerto Rico se complace en comunicar a ustedes que dará su completo respaldo a un plan de seguro médico estructurado conjuntamente por la Cruz Azul de Puerto Rico y esta Asociación, siempre y cuando que el mismo llene los siguientes requisitos:

1. Que dicho plan sea estructurado en tal forma que en un futuro no lejano pueda incluir el 80% de la población de Puerto Rico.
2. Que se cree una sub-junta de Directores compuesta de siete representantes del público y siete representantes de los médicos asociados para que entienda en todos los asuntos concernientes a los servicios médico-quirúrgicos que se presten bajo dicho plan.

3. Que los fondos correspondientes a los contratos de servicios médico-quirúrgicos sean separados de los fondos de servicios de hospitalización y que al hacer dicha separación se consigne que los mismos sean inviolables y no podrán ser usados los correspondientes a servicios médico-quirúrgicos para el pago de servicios hospitalarios y vice-versa.
4. Que dicho plan llene los requisitos de ética promulgados por la Asociación Médica Americana y por la Asociación Nacional de Escudo Azul.
5. Que se establezca un maximum de ingresos para las personas que deseen acogerse a dicho plan, y que se deje a los médicos en libertad de poder cobrar por sus servicios lo que consideren razonable a aquellos suscriptores que tengan ingresos mayores a los establecidos por el plan.

Muy atentamente,

(Fdo.) **Ricardo F. Fernández, M.D.**
Presidente

4 de noviembre de 1955

Dr. Ricardo F. Fernández, Presidente
Asociación Médica de Puerto Rico
Avenida Fernández Juncos
Parada 19
Santurce, Puerto Rico

Estimado Dr. Fernández:

Me complazco en referirme a su atenta comunicación dirigida a la Junta de Regentes de la Cruz Azul de Puerto Rico, informando que vuestra Asociación dará su completo respaldo a un Plan de Seguro Médico estructurado conjuntamente con la Cruz Azul de Puerto Rico, siempre y cuando que el mismo llenare los cinco requisitos apuntados en su referida carta.

La Junta de Regentes de esta organización, reunida en sesión ordinaria el jueves 20 de los corrientes, dió amplia y cuidadosa consideración al asunto planteado en su carta, tomando en cuenta todos los antecedentes de esta cuestión, muy principalmente el hecho incuestionable de que los Planes de Seguros Médicos, según requisitos establecidos por la organización nacional, deben funcionar con un completo respaldo de las asociaciones médicas locales, y que la ambiciosa proposición contenida en vuestra primera estipulación ha sido y es uno de los firmes e inquebrantables propósitos de la Cruz Azul de Puerto Rico.
Las distintas estipulaciones fueron consideradas separadamente, habiéndose adoptado los siguientes acuerdos con relación a las mismas:

Primera Estipulación

Se acordó aceptar esta estipulación por constituir uno de los ideales de la organización.

Segunda Estipulación

Fué acuerdo recomendar la creación de dos Sub-Juntas, una compuesta por siete representantes del Público y siete representantes de los Médicos Asociados, y la otra compuesta por siete representantes de los Hospitales y siete representantes del público, para que entiendan, respectivamente, en todos los asuntos concernientes a los servicios médico-quirúrgicos, o de hospitalización, entendiéndose que los acuerdos de dichas Sub-Juntas deberán ser sometidos a la ratificación de la Junta en pleno. Al discutirse esta cuestión se tomó en cuenta que este asunto había sido considerado por la Asamblea General de la Asociación celebrada en abril 19 y que acordó referir el asunto para su estudio y consideración de la Junta de Regentes.

Tercera Estipulación

Se acordó aceptar esta estipulación.

Cuarta Estipulación
Se acordó aprobar en principio todo lo contenido en esta estipulación.

Quinta Estipulación

Se acordó aceptar esta estipulación, disponiéndose que los límites máximos de ingresos deberán ser establecidos de mutuo acuerdo entre la Cruz Azul y la Clase Médica.

Finalmente la Junta de Regentes acordó recomendar que, a la mayor brevedad posible, debe llevarse a cabo una reunión conjunta entre representantes de la Asociación Médica y de la Cruz Azul de Puerto Rico a los efectos de sentar las bases finales para el mutuo entendimiento entre ambas organizaciones que permita lograr el objetivo que se persigue: Que la Asociación Médica de Puerto Rico dé un completo respaldo a un Plan Médico auspiciado por la Cruz Azul.

A tal efecto le ruego designe el Comité que habrá de representar a vuestra Asociación para proceder inmediatamente a hacer los arreglos necesarios para la primera reunión.

Atentamente,

(fdo.) **Arturo La Cruz**
Director Ejecutivo

Está en orden que el Comité de Escudo Azul y Cruz Azul de la Cámara y la directiva de la Asociación tengan el cambio de impresiones propuesto en esta última carta a fin de que se sienten las bases finales del programa esbozado. Esta reunión no ha podido llevarse a efecto antes debido a que en el momento en que recibimos

la anterior comunicación el doctor Colón Yordán se encontraba fuera del país.

* * *

No queremos ocupar por más tiempo la atención de ustedes relatándoles asuntos que ya son de conocimiento general por cuanto los hemos informado en nuestras cartas mensuales y en otras comunicaciones a la matrícula.

Esperamos poder seguir sirviendo a nuestra Asociación en el futuro con el mismo interés y entusiasmo que lo hemos hecho desde la presidencia, y exhortamos a todos ustedes a brindarle un respaldo incondicional a nuestro sucesor.

Muchas Gracias

Ricardo F. Fernández, M.D.
Presidente



El Dr. Ricardo F. Fernández y el Dr. Jaime F. Pou, presidente saliente y presidente electo de la A.M. P.R. cambian impresiones con los doctores Elmer Hess y George F. Lull, presidente y Secretario General de la A.M.A., respectivamente, durante su reciente visita a Puerto Rico.

INFORME DEL SECRETARIO

AÑO 1955

Señores miembros de la Cámara:

Deseamos someter a consideración de ustedes la siguiente información:

Estadística Médica:

Al 30 de noviembre de 1955 hay registrados en Puerto Rico, de conformidad con nuestros records, 1419 médicos que se dividen como se detalla a continuación:

	Socios	No socios
Dirección conocida en Puerto Rico	650	596*
En Estados Unidos y fuerzas armadas	62	77
Dirección desconocida		34
	712	707

En la edición de octubre del Boletín de la Asociación insertamos una relación de todos los médicos que aparecen en nuestros records.

Anotamos a continuación la distribución de estos médicos por pueblos y distritos:

DISTRITO OESTE

Médicos Asociados: 38 Médicos no Asociados: 28

Pueblo	Socios	No Socios	Total
Mayagüez	26	12	38
Añasco	1	1	2
Aguada	0	1	1
Cabo Rojo	3	2	5
Hormigueros	0	2	2
Lajas	1	2	3
Las Marías	0	1	1
Maricao	0	1	1
Moca	0	2	2
Rincón	0	2	2
Sabana Grande	2	1	3
San Germán	5	1	6
	38	28	66

* En esta cifra están incluídos 92 médicos que están haciendo su año de internado.

DISTRITO ESTE

Médicos Asociados: 490 Médicos no Asociados: 368

Pueblo	Socios	No Socios	Total
San Juan	307	165	472
Aguas Buenas	1	0	1
Barranquitas	0	2	2
Bayamón	21	39	60
Caguas	15	14	29
Cataño	2	3	5
Carolina	1	4	5
Canóvanas	0	4	4
Cayey	2	8	10
Ceiba	0	1	1
Comerío	1	0	1
Culebra	0	0	0
Cidra	1	1	2
Fajardo	9	25	34
Guaynabo	1	2	3
Gurabo	1	0	1
Humacao	9	7	16
Juncos	3	1	4
Las Piedras	0	1	1
Luquillo	0	3	3
Naguabo	1	1	2
Naranjito	1	1	2
Río Grande	1	4	5
Río Piedras	110	76	186
San Lorenzo	1	1	2
Trujillo Alto	0	3	3
Vieques	0	1	1
Yabucoa	1	1	2
St. Thomas	1	—	1
	490	368	858

DISTRITO SUR

Médicos Asociados: 76 Médicos no Asociados: 79

Pueblo	Socios	No Socios	Total
Ponce	53	34	87
Aibonito	1	4	5
Arroyo	1	6	7
Adjuntas	0	4	4
Coamo	2	2	4
Guánica	1	2	3
Guayama	5	6	11
Guayanilla	1	1	2
Jayuya	0	2	2
Juana Díaz	4	1	5
Maunabo	0	1	1
Patillas	0	3	3
Peñuelas	0	3	3
Orocovis	1	2	3
Salinas	2	3	5
Santa Isabel	0	2	2
Villalba	1	2	3
Yauco	4	1	5
	76	79	155

DISTRITO NORTE

Médicos Asociados: 45 Médicos no Asociados: 122

Pueblo	Socios	No Socios	Total
Arecibo	15	49	64
Aguadilla	7	24	31
Barceloneta	1	1	2
Camuy	1	5	6
Ciales	1	4	5
Corozal	1	3	4
Dorado	0	3	3
Hatillo	0	2	2
Isabela	1	4	5
Lares	0	3	3
Morovis	1	3	4
Manatí	6	3	9
Quebradillas	2	0	2
Toa Alta	0	1	1
Toa Baja	0	2	2

Pueblo	Socios	No Socios	Total
Utuado	1	5	6
San Sebastián	3	4	7
Vega Alta	2	1	3
Vega Baja	3	5	8
	45	122	167

Como informamos al principio, la matrícula actual está integrada por 712 compañeros, de los cuales 62 están en los Estados Unidos estudiando y sirviendo en las fuerzas armadas fuera del país.

Este año se unieron a nuestra matrícula los siguientes compañeros: (58 miembros regulares y 4 miembros afiliados).

Miembros regulares

Achecar Achecar, Felipe J.
Arbona de Goicoechea, José L.
Alvarez Calderón, Julián
Badillo Santiago, Ramón
Berio Suárez, Carmen L.
Biaggi Dorrego, Hipólito
Berio Suárez, Antonio
Berio Suárez, María T.
Brinz, José
Barreto Domínguez, A.
Collazo, César Augusto
Cornell, Elmer
Colberg Ríos, Herman
Castro de Suárez, Carmen
Cora Andrade, Marina E.
Díaz de Garau, Priscila
Feliciano Rodríguez, Héctor A.
Fuentes Cruz, Claude E.
Fernández Sariego, Gilberto
Fortuño, Roberto F.
Frasqueri Toste, Eduardo R.
García Lascot, Eulalio
Gómez Acevedo, Manuel
Garriga Prida, José E.
Hidalgo Cestero, Carlos A.
Isales, Ramón
Javier de Young, Haydeé
López Elías, Francisco
López Vargas, Fernando E.
Lugo, Alberto E.
Moore, Charles B.
Morales Boyer, Rafael
Mella, Juan Luis
Muñoz, Aurea Isabel
Martínez de Polo, Hilda
Montalvo Durand, Eladio A.
Negrete Añil, César A.
Nine Curt, José
Núñez Colón, Neida
Ortega Guzmán, Francisco A.
Pérez Lara, Rafael A.
Prado, José del
Piovanetti Bonelli, Simón
Quiñones Acosta, Francisco
Remus Quintana, Luis E.
Ríos Pagán, Julián
Ramírez Kohl, Emilio
Rivera Cintrón, Fco. José
Ramírez Weiser, Rafael R.
Sampayo, Héctor Manuel
Suárez Alvarez, José F.
Sepúlveda, Celestino E.

Sánchez Longo, Luis P.
Torres Machin, Arturo
Toro Duncan, Luis del
Von Micsky, Lajos I.
Wurdemann, Alma Louise
Zeni, Celso Vittorio

Miembros Afiliados

Bosch Faura, José E.
Frank, Julio E.
Rubio Herrera, Luis A.
Rigau Marques, José M.

Solicitudes regulares pendientes de consideración:

Oscar A. Ruiz Sosa
Waldo J. Rodríguez
Rafael O. Fernández
Raimundo W. Rodríguez

Solicitudes de miembros afiliados por considerar:

Rafael Guerrero Guerrero
Pedro Luis Nieves López
Zenobio R. Martínez Cancel
Jack Shepard

Las bajas habidas en nuestra matrícula este año han sido:

Por muerte:

Dr. Hilario Caso
Dr. Alvaro Santaella
Dr. Pedro Conde
Dr. Federico Velázquez
Dr. Fernando de Juan
Dr. Manuel Quevedo Báez

Por haberse ausentado para Estados Unidos

Dr. Celso Ramón García

Por no pagar la cuota

Dr. Plácido Arrache

Reuniones Administrativas

Este año se han celebrado por los directores y comités administrativos de la Asociación un total de 61 reuniones. Un detalle de estas reuniones se incluye al final de este informe.

Reuniones Científicas

También incluímos al final una relación de todos los actos científicos celebrados durante el año. El total de conferencias

dictadas monta a 63. Estos actos fueron auspiciados por los siguientes grupos:

Comité Científico
Sección de Oftalmología y Otorrinolaringología
Sección de Urología
American College of Physicians
Sección de Pediatría
Asoc. Médica Distrito Oeste
Sección de Medicina Interna
Sección de Cirugía General
Sección de Obstetricia y Ginecología
Asociación Médica del Distrito Sur
Junta de Cursos Postgraduados
Asoc. Médica Distrito Este

Durante la asamblea que dará comienzo el martes se dictarán 55 conferencias adicionales, lo que trae el programa de todo el año a un total de 118 ponencias.

Asociación Médica Americana

Es con verdadera satisfacción que informamos el hecho de que nuestra matrícula ha respondido bastante bien a nuestra exhortación para que el mayor número se acoja a la Asociación Médica Americana. Al 30 de noviembre tenemos un total de 182 compañeros que son miembros activos de la Asociación Médica Americana. Una lista de estos colegas aparece más adelante en nuestro informe.

Al revisar el informe del Secretario de la Asociación Médica Americana de fecha 29 de noviembre de 1955 encontramos que él comunica a la Cámara de Delegados de la A.M.A. que en Puerto Rico hay 225 miembros activos. Hemos solicitado una relación de éstos para revisar nuestros records.

Actividades generales

Como de costumbre, hemos realizado cuantas gestiones caen dentro de nuestro radio de acción; hemos suplido a los socios las credenciales y certificaciones correspondientes; hemos colaborado con los demás compañeros de directiva en todo cuanto ha estado a nuestro alcance y hemos enviado a la Asociación Médica Americana toda la información solicitada.

Cordialmente,

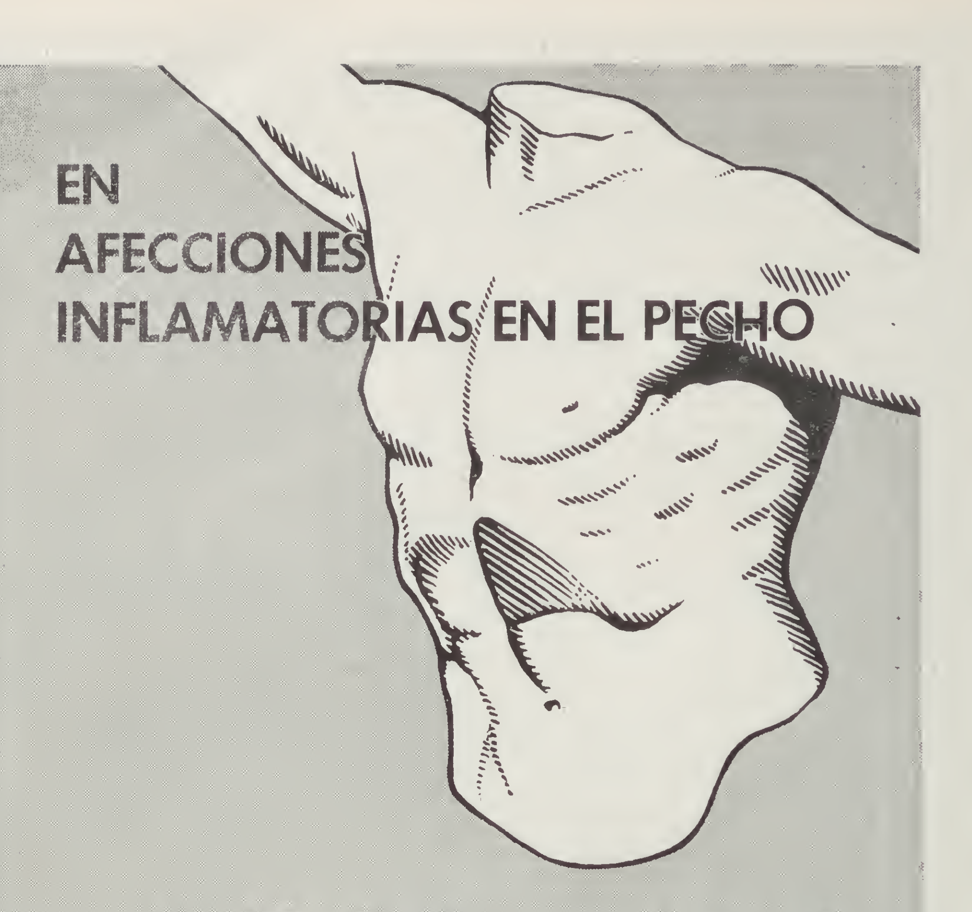
Luis R. Guzmán López, M.D,
Secretario



FOTOGRAFIA HISTORICA (Cortesía del Lcdo. A. F. Legrand).

PROFESORES DEL INSTITUTO UNIVERSITARIO JOSE DE DIEGO, SAN JUAN, P. R. - ABRIL DE 1916.

Entre otros hemos podido identificar a: Dr. M. Quevedo Báez, Sra. Dr. R. M. Suárez, José de Diego, J. Federico Legrand, Vicente Balbás Capó, Rafael López Landrón, Ignacio López Mergeliza, Pedro Timothée, Manuel Rodríguez Serra, Jaime Annexy, Don Teodoro Aguilar y Lcdo. Juan M. Herrera.



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BOLETIN
DE LA
ASOCIACION MEDICA
DE
PUERTO RICO

Organo Oficial de la Asociación Médica de Puerto Rico

Enero-Diciembre, 1955
Vol. 47

San Juan
Imprenta Venezuela
1955

LISTA DE COLABORADORES AL VOLUMEN 47 DEL

BOLETIN DE LA ASOCIACION MEDICA DE PUERTO RICO

AXTMAYER, ALFRED L., San Juan
BASORA DEFILLO, J., Santurce
CASANOVA DIAZ, A. S., Santurce
COLON RIVERA, EGIDIO S., Santurce
COSTA MANDRY, OSCAR, Santurce
COSTAS DURIEUX, J. L., Ponce
CRUZ HERNANDEZ, HECTOR, Arecibo
CURBELO, PABLO, Santurce
EHRLICH, LASZLO, San Juan
FERNANDEZ, RICARDO F., San Juan
FERNANDEZ CERRA, E., Hato Rey
FLAX, HERMAN J., Santurce
GUZMAN LOPEZ, LUIS R., Santurce
HADDOCK SUAREZ, JENARO, Santurce
HEINMAN, HAROLD, Santurce
JESUS, JOSE A. de, Santurce
JIMENEZ TORRES, CARLOS F., Ponce
MENDOZA MENDOZA, PEDRO, Aguadilla
OLAVARRIETA, SARA T., Santurce
O'NEILL, EDWARD, San Juan
PADRO, FERNANDO, Santurce
PASSALACQUA, LUIS A., Santurce
PANIAGUA, MANUEL E., Santurce
PICO, GUILLERMO, Santurce
QUINTERO, ERNESTO, Santurce
RIVERA, JULIO V., San Juan
RODRIGUEZ, HECTOR F., Ponce
RODRIGUEZ OLMO, J., Arecibo
RODRIGUEZ PEREZ, DAVID, Río Piedras
ROSENFIELD, A. B., Minnesota
RULLAN, ANTONIO, Santurce
SANABRIA, JOSE F., Santurce
SIFONTES, JOSE E., Río Piedras
SIFRE, RAMON A., Santurce
STREET, DANA M., Memphis, Tenn.
SUAREZ, RAMON M., Santurce
SUAREZ BENITEZ, RAMON M., Santurce
SUSONI, ANTONIO H., Arecibo
TAINTER, MAURICE L., Rensselaer, N. Y
YORDAN, EDGARDO, Ponce

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